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THE RELATIONSHIP OF SMOKING AND CANCER OF THE LUNG

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New Orleans, La.

Undoubtedly many lay persons as well as physicians have been concerned by the increased incidence of cancer generally. The increase in most cancers is due to the fact that cancer primarily is a disease of older persons and because at present life expectancy is considerably greater than 25 years ago; there are more persons living to a cancer age than ever before. In fact, of all persons 90 years of age a greater percentage will develop cancer than those 80; of those 80 years of age a greater percentage will develop cancer than those 70, and so on. This is true of every type of cancer except one—cancer of the lung—which has increased and is increasing more than any other cancer in the body.

Whereas cancer of the lung occurs primarily in men (at the present time about 90 per cent of cancers of the lung occur in males), it is increasing in both sexes. According to the Public Health statistics cancer of the lung increased in women from 2.3 per 100,000 population in 1933 to 4.7 per 100,000 population in 1948 (fig. 1). The respective increases in men in these two periods was from 4.6 to 17.8 (fig. 2). If one considers the increase after age standardization, cancer of the lung increased in women from 0.6 per 100,000 population in 1914 to 4.3 per 100,000 population in 1950 (fig. 3); whereas, in men, the increase was from 0.7 per 100,000 population in 1914 to 19.6 per 100,000 population in 1950 (fig. 4). There was an increase of 411 per cent in the incidence of cancer of the lung in men from 1930 to 1948 (fig. 5). The deaths from lung cancer in both sexes increased from 3,900 in 1930 to 27,000 in 1953. Clemmesen,[2] in a recent communication, believes that bronchogenic cancer is increasing so rapidly that it can be expected that in men it will equal in frequency all other cancers combined. He refers to the increase in the incidence of bronchogenic cancer as being *pandemic*.

DEATH RATES FOR SELECTED RESPIRATORY DISEASES AND SITES OF CANCER
AMONG WHITE FEMALES, United States, 1933-1948
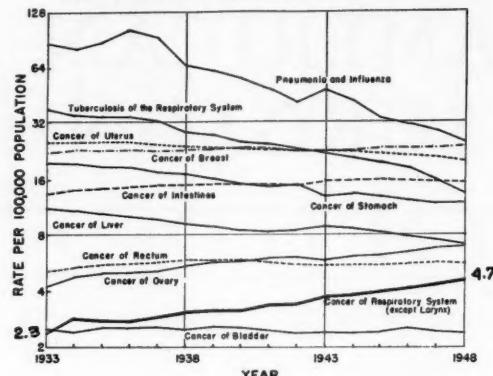
FIG. 1

DEATH RATES FOR SELECTED RESPIRATORY DISEASES AND SITES OF CANCER
AMONG WHITE MALES, United States, 1933-1948
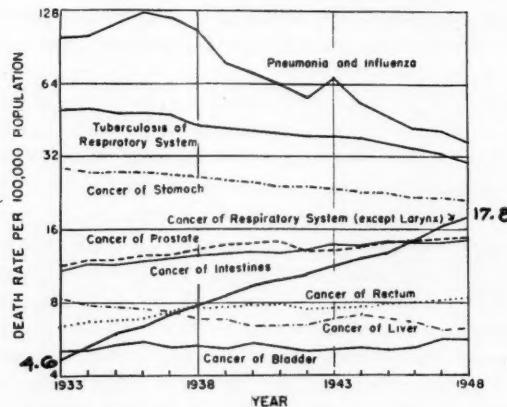
ª Standardized for age on the 1940 United States population

SOURCE: NATIONAL OFFICE OF VITAL STATISTICS   STATISTICAL RESEARCH SECTION
                                                  AMERICAN CANCER SOCIETY 9-53

FIG. 2

Although, as stated previously, cancer is increasing generally, cancer in females
has actually decreased. This is a real tribute to the educational program of the
American Cancer Society and its efficacy in that women have learned that it
pays to consult their physicians regularly with the result that precancerous
lesions are being detected and corrected, producing a decrease in the incidence
of cancer in women. On the other hand, cancer in men has increased very re-
markably in the past decade and this has been due almost entirely to the tremen-

MALE- LUNG CANCER DEATHS
IN UNITED STATES

FEMALE-LUNG CANCER DEATHS
IN UNITED STATES

FIG. 3

FIG. 4

DEATHS FROM CANCER OF LUNG
IN WHITE MALES IN UNITED STATES
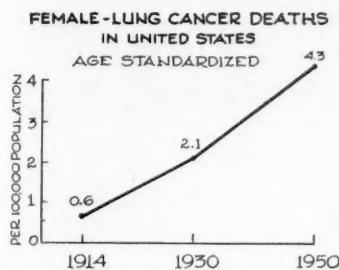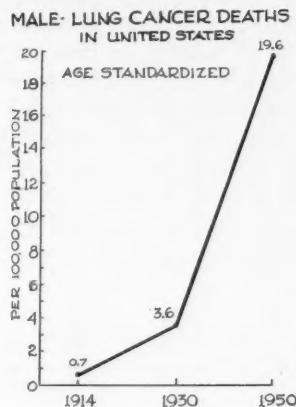
HAMMOND 1954
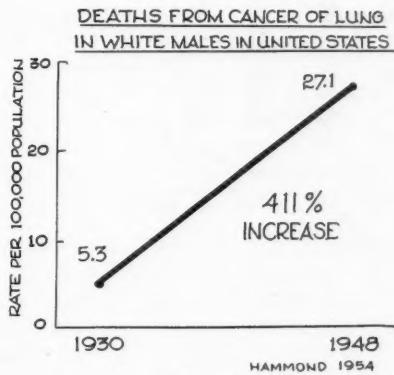
FIG. 5

dous increase in the incidence of bronchogenic cancer. If one excludes broncho-
genic cancer, there has been very little increase in the incidence of cancer in men
in the past quarter of a century (fig. 6).

There are many who maintain that cancer of the lung is not increasing but
that it is simply being diagnosed at the present time, whereas previously it was
not recognized and the condition was diagnosed as some other lesion. If this were
the case, the one lesion with which cancer of the lung might be confused would
be tuberculosis. Whereas there has been a general decrease in the incidence of
tuberculosis in recent years, there has been no decrease in the incidence of
tuberculosis in the older age group in which cancer of the lung occurs. In fact,
if the increase in the incidence of cancer of the lung were due to better diagnosis
at the present time, at the same time there should be a decrease in the incidence
of tuberculosis in the older age group commensurate with the increase in the

CANCER DEATH RATES' AMONG WHITE MALES AND FEMALES
United States, 1933-1948
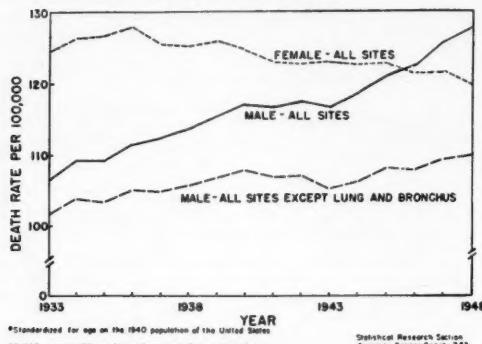
*Standardized for age on the 1940 population of the United States
SOURCE National Office of Vital Statistics; Bureau of the Census

Statistical Research Section
American Cancer Society, 7-53

FIG. 6

RESPIRATORY SYSTEM EXCEPT LARYNX CANCER
AND RESPIRATORY SYSTEM TUBERCULOSIS -
DEATH RATES AMONG WHITE MALES, AGES 65-69
UNITED STATES-1933-1948
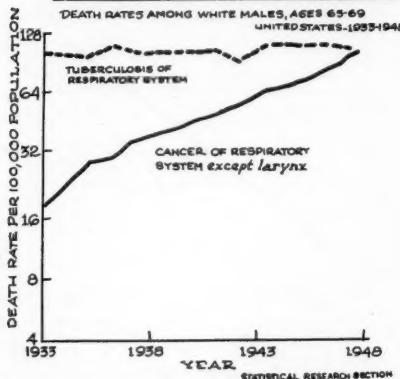
STATISTICAL RESEARCH SECTION
AMERICAN CANCER SOCIETY-9-93

FIG. 7

incidence of cancer of the lung, but such is not the case (fig. 7). Another reason why I am convinced that there has been an actual increase in the incidence of bronchogenic cancer and not a relative one due to more correct diagnoses, is that in those countries where autopsies have been done routinely for many years, such as the Germanic countries, bronchogenic cancer has increased as it has in this country, and it is inconceivable that the well-trained, meticulous German pathologist would miss bronchogenic cancer at autopsy.

In the United States, cancer of the lung in 1920 represented 1.1 per cent of all cancers; in 1930, 2.2 per cent of all cancers; in 1948, 8.3 per cent of all cancers. Upon the basis of this increase from 1920 to 1948 and because of the presence of certain carcinogenic factors which will be discussed later, we have ventured to

predict that in 1970 cancer of the lung will represent 18 per cent of all cancers or approximately 1 in every 5 (fig. 8). If one considers only males, in whom cancer of the lung is much more likely to occur than in females, it can be predicted that in 1970, unless something is done to prevent it, 1 of every 1 or 2 men with cancer will have a cancer of the lung. It is estimated by the American Cancer Society that 1 of every 4 persons living will develop a cancer of some type. It therefore can be predicted that in 1970, unless something is done to prevent it, 1 of every 4 or 8 men living in the United States will develop cancer of the lung. Cancer of the lung has increased not only in the United States but has increased in other countries. In Holland, from 1924 to 1951, cancer of the lung increased in women
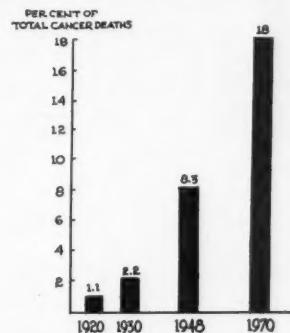
Fig. 8. Increase in the deaths from cancer of the lung in the United States from 1920 to 1948 with a predicted incidence in 1970.

tenfold and in men twenty-four fold (fig. 9).[5] It has likewise increased in the British Isles. In 1931, cancer of the lung represented 0.5 per cent of all deaths, whereas in 1952 it represented 5 per cent of all deaths. In 1931, cancer of the lung represented 5 per cent of all cancer deaths and in 1952 it represented 26 per cent of all cancer deaths (fig. 10). In fact, cancer of the lung, in England, is by far the most common cancer. In 1950, 10 per cent of all men who died between the ages of 45 and 55 died of primary cancer of the lung—an appalling fact![3]

As mentioned previously, cancer of the lung is the one cancer in which the incidence does not correspond to the pattern followed by other cancers. Whereas all other cancers increase with advancing age past 50, cancer of the lung increases precipitously to reach a peak age at the present time of 55 years following which there is a rapid decline in incidence (fig. 11). This exceptional behavior of cancer of the lung is not new; in fact, 10 years ago, the age at which the peak incidence occurred was 65 but the peak was not as high as it is at the present time. Five years ago, the peak age was 60 and the height of the peak was somewhere between that which was present 5 years ago and that at the present time (fig. 12). The lack of conformity of the lung cancer increase to the pattern followed by all other cancers is due to a definite reason. For many years I have been convinced that the unprecedented increase in the incidence of cancer of the lung is due to the carcinogenic effect of cigarette smoke, and I am sure that this accounts for the

LUNG CANCER DEATHS IN HOLLAND

FIG. 9

LUNG CANCER DEATHS IN MALES IN ENGLAND AND WALES

FIG. 10

AUTOPSY INCIDENCE

FIG. 11

BRONCHOGENIC CARCINOMA
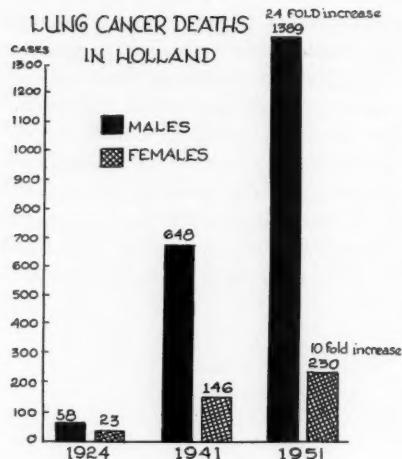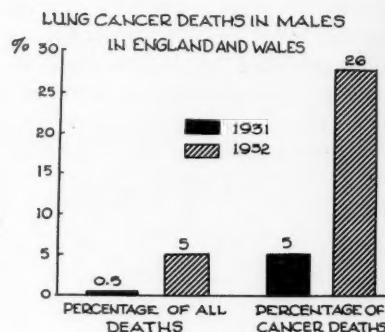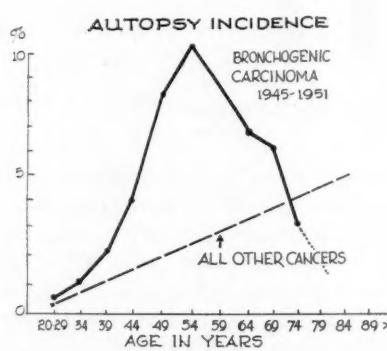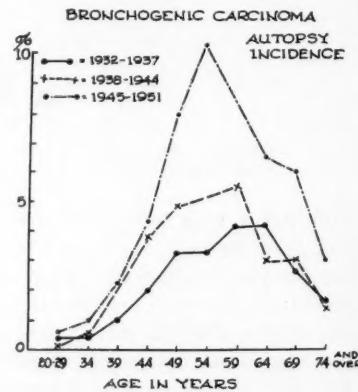
FIG. 12

peculiar behavior in the pattern followed by cancer of the lung. The tremendous increase in the incidence of cancer of the lung is due to the fact that cigarette smoking has increased enormously. The peak age is becoming higher and at an earlier age, because more people are smoking heavily and beginning to smoke at an earlier age and, therefore, the carcinogenic effect is being produced at an earlier age today than it was 10 years ago and an even earlier age than it was 5 years ago. The fall-off in the incidence of cancer of the lung after the peak age is obtained is due to the detrimental effect of the tobacco on the heart and blood vessels of individuals who have smoked heavily, producing coronary heart disease resulting in the death of the individual before the carcinogenic effect has had a chance to exert itself. It is a well known fact that cigarette smoking produces cardiovascular damage.

The annual consumption of cigarettes per capita in the United States has in-increased tremendously (fig. 13); in fact, in persons 15 years of age and older the number increased from 630 in 1920 to 3,500 in 1953 (fig. 14). There also is a distinct parallelism between the consumption of cigarettes in the United States and the increase in the incidence of bronchogenic cancer (fig. 15).

It is thoroughly understandable that there are many individuals who are unwilling to accept a causal relationship between smoking and bronchogenic cancer. Obviously those concerned with the raising of tobacco, its marketing, the processing and the sale of the final product are reluctant to accept any such causal relationship. In addition, the cigarette user who has become addicted to its use, even though he might be medically trained, is reluctant to admit that anything that he does might be harmful. Because of this there are many who will not accept the presently available evidence of the causal relationship between smoking and lung cancer. The statement frequently is made that there can be no causal
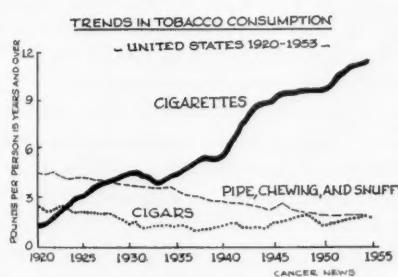
Fig. 13
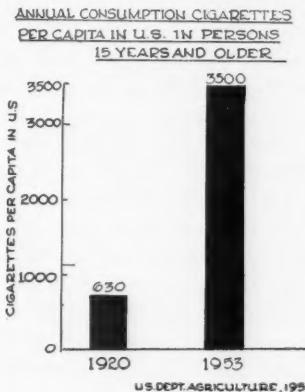
Fig. 14
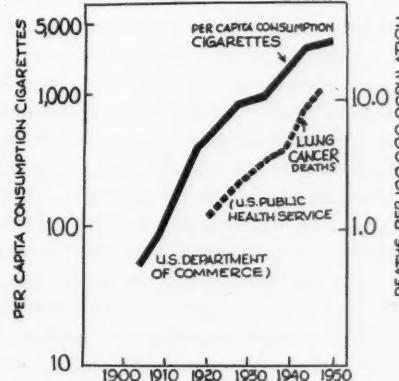
Fig. 15

relationship between smoking and lung cancer because, if there were, there should be a commensurate increase in the fatality incidence from cancer of the larynx which is not the case. Whereas it is true that the death rate from cancer of the larynx has not been proportionate to the death rate from cancer of the lung, it is equally as true that cancer of the larynx has been and is increasing with the same frequency as cancer of the lung. The difference between cancer of the larynx and cancer of the lung is that cancer of the larynx is readily diagnosed, usually when the lesion is early, and can be very satisfactorily treated with cure in a large percentage of instances, whereas cancer of the lung is difficult to diagnose early and because of this the death rate from cancer of the lung has been high. At one time, the death rate from appendicitis was high, but no fair minded individual would hazard a statement that appendicitis has decreased or is not a common disease at the present time because few cases are found at autopsy.

Another half truth which frequently is presented against a possible causal relationship between smoking and cancer of the lung is that cancer of the lung is more frequent in England than it is in the United States although more cigarettes are consumed in the United States than in England. This statement, even though true, is only a half truth, because, although more cigarettes are consumed in the United States at the present time than in England, such has been the case only during the last 8 years. Prior to 8 years ago the British people smoked a great deal more than we, and they are now paying the price for this heavy consumption of tobacco. In another 15 or 20 years the incidence of lung cancer in the United States will be considerably more than it is in England at the present time, because we smoke a great deal more than they. The tragic significance of this is evident when one recalls that in England over 25 per cent of all the cancer deaths in males are due to lung cancer and that in 1950 10 per cent of *all* deaths in males between the ages of 45 and 55 were due to this disease. It has been suggested that the increased incidence of cancer of the lung is due to carcinogens contained within the *smog* which is quite prevalent in England. That this cannot be a factor is illustrated by the fact that across the English Channel in Denmark, where the incidence of cancer of the lung has increased as it has in England and is commensurate with smoking habits, there is no *smog* whatsoever.

Several years ago Graham and Wynder[3] performed an experiment which proved without any question of a doubt that a carcinogen exists in the smoke from cigarettes. Employing a robot machine which smokes 24 cigarettes almost identically to the manner in which humans smoke, in that every 60 seconds a *drag* of 2 seconds is taken, they collected the smoke from cigarettes and cooled it. The *tarred* residue was placed in a solvent and three times a week it was applied to the skin surfaces of animals. In a control group, the solvent without the *tarred* residue was used. At the end of 8 months, 1 benign tumor developed at the site of the application of the *tarred* residue. At the end of a year 1 real cancer developed and at the end of 2 years 44 per cent of the animals developed a cancer at the site of the application of the *tarred* residue. The cancer was identical with human cancer histologically, it metastasized and produced the death of the animal and was in many instances transplantable. In none of the controls in

which the solvent alone was applied to skin of animals did either a benign or malignant tumor develop. The statement frequently has been made that one cannot compare animal and human cancer. In this investigation no attempt was made to make this comparison. The investigation simply proves that contained within the smoke from cigarettes is a carcinogen. Since it is an established fact that cancer of the lung is increasing more than any other cancer in the body and also that there is a parallelism between the consumption of cigarettes in the United States and the incidence of cancer of the lung and because the investigation of Graham and Wynder has shown that there is a carcinogen in the smoke from cigarettes, it is a perfectly logical conclusion that the cause for the unprec-

The Trachea, Coryna And Bronchi
Showing The Areas Taken For Histological
Study.

Posterior View
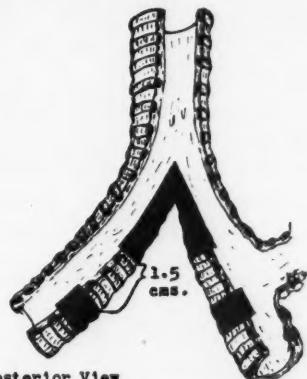
FIG. 16. Diagram showing areas of the bronchi from which sections were taken for histologic examination as illustrated by the solid black. At autopsy the coryna was removed and portions of the mucous membrane, 1.5 cm. distally in each main stem bronchus.

edented increase in the incidence of cancer of the lung is cigarette smoking, establishing the causal relationship between smoking and cancer of the lung.

Another antagonistic argument which frequently is suggested is that precancerous lesions are not found in the respiratory tract of smokers. After consulting with pathologists, I learned that it is exceptional for pathologists to look for precancerous lesions in the bronchi in doing autopsies. Approximately 2 years ago the Department of Pathology at Tulane became interested in this problem and began looking for possible precancerous lesions in the blocks of bronchial wall removed from the region of the coryna and also from each main bronchus of individuals who came to autopsy (fig. 16). They found that there were changes in the bronchi which on microscopic section varied according to the amount smoked. The individual who had never smoked had a normal bronchial mucous membrane on microscopic examination (fig. 17). The moderate smoker had changes which consisted of metaplasia (fig. 18), and the heavy smoker had
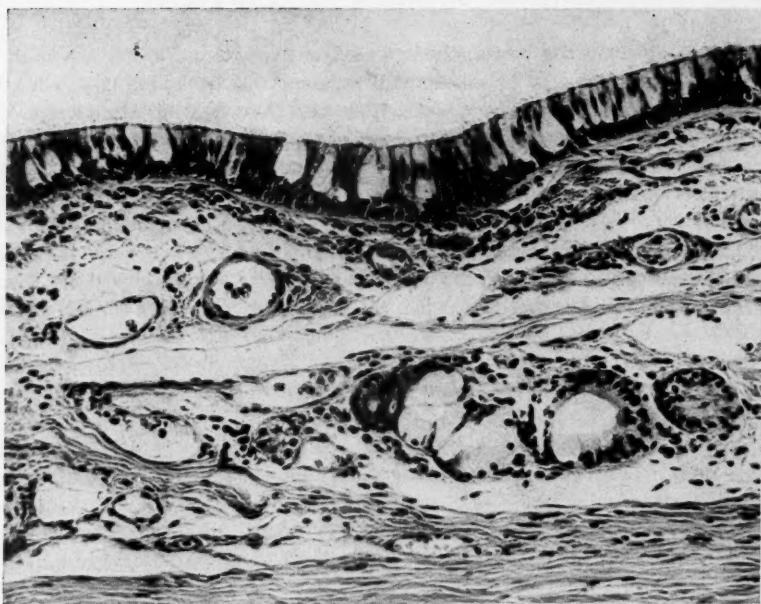
Fig. 17. Bronchial mucous membrane of a man in his early fifties who had never smoked.

Fig. 18. Bronchial mucous membrane of a man in his early fifties who had smoked moderately. In contrast to the mucous membrane in figure 17, there is definite metaplasia in the mucosa.
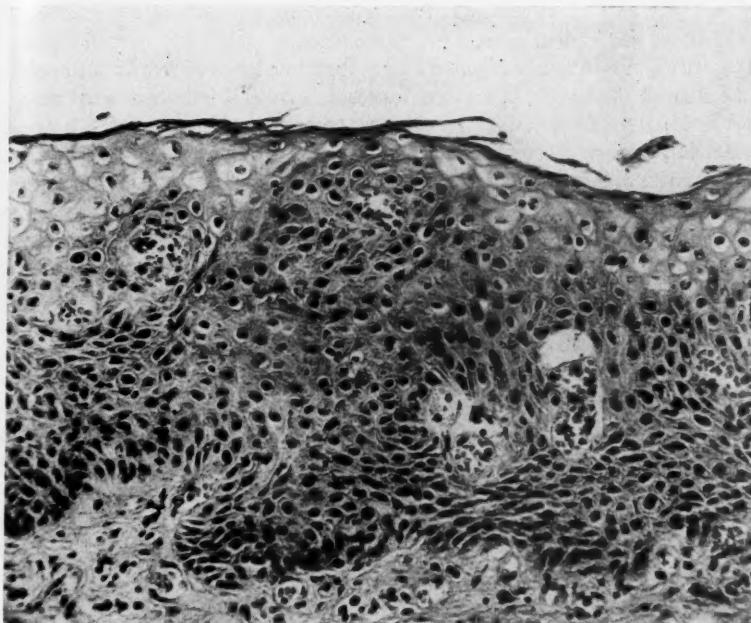
Fig. 19. Mucous membrane of a man in his early fifties who had smoked heavily. There is tremendous metaplasia, so severe that it can be considered as a precancerous lesion.

marked changes which were interpreted as premalignant lesions (fig. 19). The only reason patients with premalignant lesions did not develop cancer of the lung was that they died of something else before their lung cancer developed. This study demonstrates that cigarette smoking is associated with precancerous lesions in the bronchial mucosa and that the degree of metaplasia increases with the amount smoked.

For many years those who have been concerned with the treatment of patients with bronchogenic cancer have been impressed with the high frequency of heavy smokers among men who have this disease. In fact I have been so impressed by this that for the past 5 years I have categorically made the statement that if any man who has a pulmonary condition which might be bronchogenic cancer, if he is not a smoker, the lesion is not an epidermoid cancer but is either an adeno-carcinoma, which is not produced by a carcinogen, or is an inflammatory lesion. In 5 years time I have been wrong only once. The positive history of smoking at present is our best method of making a diagnosis of a malignant lesion of the lung. Well over 95 per cent of men with cancer of the lung are cigarette smokers as contrasted with a control group of from 65 to 75 per cent without cancer of the lung. Recently, Breslow and associates[1] showed, on the basis of careful examination of 518 patients with bronchogenic cancer as compared with 518 patients of the same age and sex without lung cancer that in men over 50 years of age,

lung cancer was 4 to 11 times as frequent among smokers as nonsmokers and 7 to 27 times as frequent among heavy smokers.

The criticism frequently is made that a high incidence of smokers in patients with cancer of the lung is of no significance, but until it is known what the incidence of lung cancer is among smokers as compared to nonsmokers, a possible relationship cannot be considered. The answer to this question was not available until the preliminary report of the investigation by the American Cancer Society, which was made before the American Medical Association in San Francisco in June, 1954.[4] The survey conducted by the American Cancer Society briefly was as follows: Twenty-two thousand volunteer workers in urban areas, in highly industrialized areas, and in rural areas interviewed approximately 200,000 men between the ages of 50 and 70, because this is the age group in which cancer of the lung occurs most frequently. A very elaborate questionnaire was filled out concerning the man's smoking history. One year later the interviewer reinterviewed each of the men whom she had interviewed the year before. In the meantime some of the men interviewed had died. A photostat of the death certificate was secured and, if the individual had died of cancer and had had a biopsy or autopsy, an attempt was made to obtain the microscopic slide of the tissue removed. Two years after the original interview, a third interview was secured. Approximately 5,000 men had died since the beginning of the survey, representing 2.6 per cent of the entire group interviewed. The results of this investigation are extremely illuminating. The investigation showed that there has been a tremendous increase in the incidence of smoking in recent years. Of the men in the age group 65 to 70, 21.6 per cent had never smoked and 20.3 per cent were heavy smokers; whereas in the age group 50 to 55 only 15 per cent had never smoked and 43 per cent were heavy smokers (fig. 20). The study showed that there was a definite increase in the death rate from all causes among the individuals who smoked cigarettes as compared with those
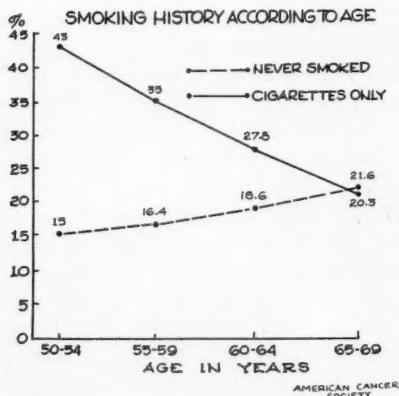
FIG. 20. Diagram showing that men in the fifty to fifty-five age group smoke much more than men in the sixty-five to seventy year age group.
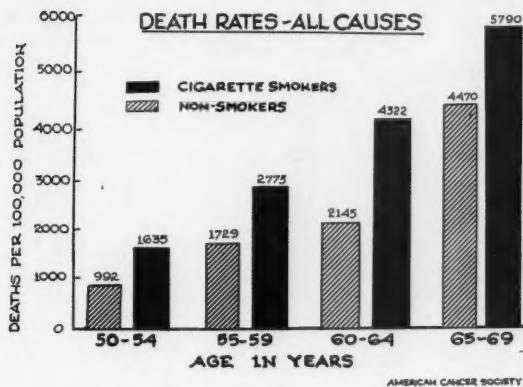
FIG. 21
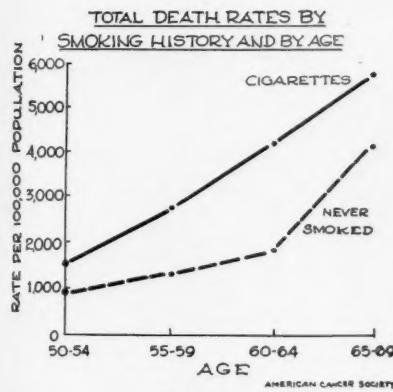
FIG. 22
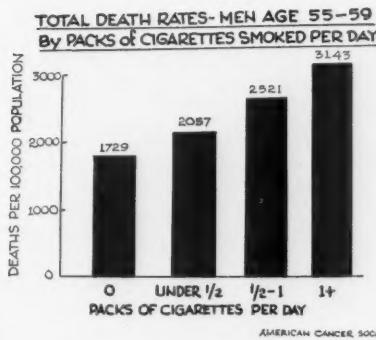
FIG. 23

who did not smoke; and this was true in all the age groups and the death rate varied according to the amount smoked (figs. 21, 22, and 23). There was considerable difference in the death rate from cancer in all sites among cigarette smokers and nonsmokers (figs. 24 and 25). This difference was particularly great in the age groups 60 to 65 and 65 to 70 in which the death rate was over twice as great among the smokers as compared with nonsmokers (fig. 25). Although the death rate from cancer of the lung was particularly higher among smokers as compared with nonsmokers, it was found that the death rate from other cancer besides cancer of the lung also was greater among cigarette smokers than among nonsmokers (fig. 26). An incidental finding was that the death rate from coronary disease was definitely higher in cigarette smokers than in nonsmokers and this was particularly great in the 60 to 65 age group (figs. 27 and 28). This was not unanticipated, because many years ago Raymond Pearl[7] showed
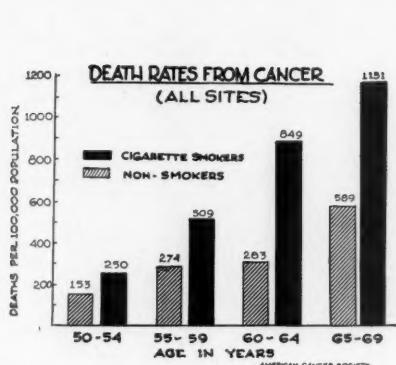
FIG. 24
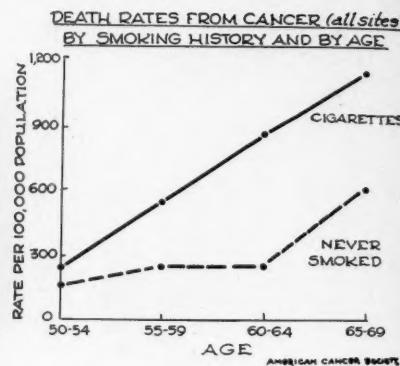
FIG. 25
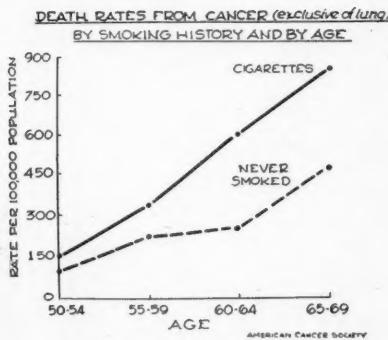
FIG. 26

that the survival rate of cigarette smokers was definitely less than among non-smokers (fig. 29). Also, the American Cancer Society study[4] showed that the death rate from coronary disease was proportionate to the amount smoked. Of the group that smoked none at all there were 689 per 100,000 population deaths; of those who smoked less than a half pack there were 769; of those who smoked from a half a pack to a pack there were 1,287, and of those who smoked more than a pack there were 1,477 (fig. 30). To recapitulate, the study showed that among heavy cigarette smokers the over-all death rate was 75 per cent higher than among nonsmokers. The death rate from heart disease was 95 per cent higher than among nonsmokers; the death rate from all cancers was 156 per cent higher than among nonsmokers, and the death rate from lung cancer was 400 per cent higher than among nonsmokers (fig. 31)!

The study by the American Cancer Society[4] which was made in a completely impartial manner and which includes a large enough group of cases to be statistically significant proves without any reasonable question of doubt that there is a causal relationship between smoking and cancer—particularly lung cancer—as well as a causal relationship between cigarette smoking and heart disease. In

DEATH RATES FROM CORONARY DISEASE

FIG. 27

DEATH RATES FROM CORONARY ARTERY DISEASE
BY SMOKING HISTORY AND BY AGE

FIG. 28

PEARL'S LIFE TABLE
SURVIVORSHIP OF NON-USERS OF TOBACCO
VERSUS HEAVY SMOKERS

FIG. 29

DEATH RATES FROM CORONARY DISEASE
MEN AGE 55-59

FIG. 30

% INCREASE OF DEATHS IN HEAVY
CIGARETTE SMOKERS OVER NON-SMOKERS
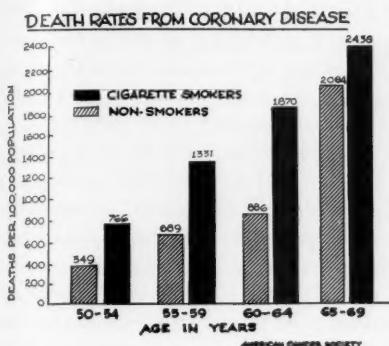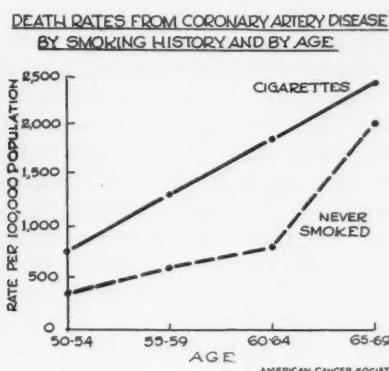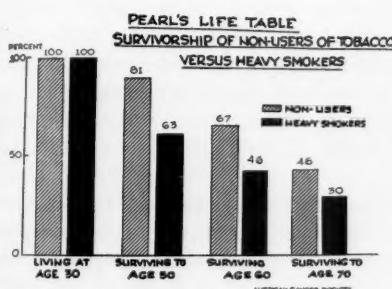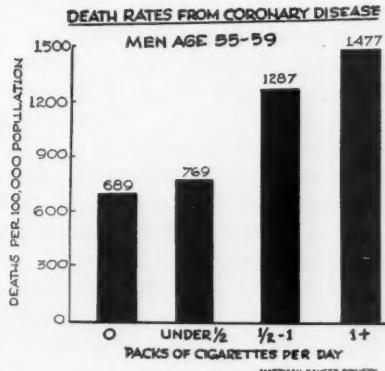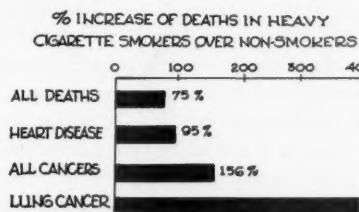
FIG. 31

spite of this there are many who are reluctant to accept this relationship. Clemmesen,[2] an eminent Danish pathologist, who has been very much interested in epidemiology of cancer for years, states: "It is true that we have no warrant of the effect, but where were the warrants in the combat against epidemics in the past? Let no one believe that the attitude of the public will remain indifferent when in one or two decades the extent of the catastrophe will become apparent

to everyone." If there were as much evidence that the Brooklyn Bridge were unsafe for use, unquestionably it would be closed to traffic immediately until its safety could be determined. In addition to those who are unwilling to accept the causal relationship between cigarette smoking and cancer, there are those who take a fatalistic attitude and believe that, because they have smoked for 20 years or more, the die is cast and nothing can be done to prevent their developing cancer. Although such probably is true in some instances, undoubtedly there are many in whom the process is reversible, and by discontinuing smoking they will prevent the development of the true malignant lesion. Fortunately, individuals vary in susceptibility to the development of malignant lesions and whereas it is conceivable that the individual who has a greater susceptibility will develop bronchogenic cancer after using cigarettes for 15 or 20 years, there are others who might continue for longer periods of time and still be immune. On the other hand, I believe that if one lives long enough and continues smoking he will ultimately develop bronchogenic cancer.

Because of the pandemic increase in the incidence of bronchogenic cancer, it is essential that the profession urge discontinuance of smoking and that young individuals should be dissuaded from acquiring the habit. Clemmesen[2] states that unless young persons are prevented from acquiring smoking habits, we will not be able to avoid *one of the major catastrophes in medical history*. In England the British Committee on Cancer and Radiotherapy[6] reported to the House of Commons that "young people should be warned of the risks apparently attendant on excessive smoking" and following this Ian Macleod, British Health Minister, reiterated to the youth of England the danger of smoking and its relationship to cancer of the lung.

Unfortunately, bronchogenic cancer in its incipiency produces few manifestations. The individual who is a heavy smoker usually has a cough, the so-called *smoker's cough*, and because of this, a cough is likely to be disregarded. It is imperative, however, because of the definite carcinogenic effect of cigarette smoking that men who smoke heavily, whether they are members of the profession or lay people, should have a roentgenogram of the chest at least every 6 months or preferably every 3 months so that when a bronchogenic cancer does develop it can be detected at the time while it is still relatively small. Need for such precaution is emphasized by the fact that in our series of over 1,500 cases of bronchogenic cancer resection was possible in only 32 per cent and in only 10 per cent was there no gross evidence of extension beyond the lung.

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BLOOD VOLUME STUDIES WITH THE USE OF I¹³¹ TAGGED ALBUMIN

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With the introduction of each new laboratory method for clinical assay, the clinician must acquaint himself with its applicability, its significance and its usefulness. The technic for estimating blood volume now is a fairly accurate procedure which can be done quickly and cheaply with the aid of radioactive isotopes. The introduction of the well-type scintillation counter has greatly simplified the use of gamma-emitting isotopes for blood volume determination, and has enabled one to use such small quantities of radioactivity that the health hazard to the patient and administering personnel is negligible.

Blood volume currently is being evaluated in many hospitals but its status still is not generally recognized as a necessary clinical adjunct. Accumulating experience tends to indicate that a knowledge of the blood volume is more useful, especially in surgery, than is a knowledge of the red cell count and the hemoglobin. We have nothing new to add to the subject of blood volume *per se*. The object here is only to evaluate routine blood volume determinations in a general hospital, particularly with reference to surgical patients, and to suggest a technic for blood volume determination.

TECHNIC

A simple method of blood volume determination is by the dilution technic with the intravenous injection of radioactive iodinated human serum albumin (IHSA)*. It has advantages over the use of Evans blue T-1824 in that the measurement of radioactivity is more precise than the measurement of color and in that it can be repeated at short intervals. Red cells tagged with P³² or with radioactive chromium as $Na_2Cr^{51}O_4$ give a more accurate method of estimating red cell mass but are not a more accurate estimation of total blood volume. Both methods, however, entail incubation of the patient's blood with the radioactive sodium chromate or sodium phosphate and thus do not lend themselves to the simple routine use especially required in surgical emergencies.

Gray and Frank[19] have described a method of determining plasma volume by

injecting radioactive chromic chloride directly into the blood stream. They found that the radioactive chromic chloride is rapidly bound to the plasma proteins *in vivo* in a stable union. The material could be used in an identical manner as is iodinated serum albumin, so that the process of tagging albumin with I^{131} would be eliminated.

The technic used in our determinations varied only slightly in the institutions in which these studies were effected:

1. A sterile isotonic salt solution, from 5 cc. to 20 cc., containing approximately 3 microcuries of iodinated human serum albumin is injected intravenously into an antecubital vein.

2. A standard is prepared by measuring an identical quantity of the IHSA solution in the same syringe and emptying it into a volumetric flask containing 2000 ml. of isotonic salt solution. Ten minutes after the injection, 3 cc. or 5 cc. of blood are withdrawn from the centrolateral antecubital vein into a heparinized syringe and emptied into a disposable plastic tube or into a graduated test tube.

3. The activity is counted immediately in a well-type scintillation counter.

4. A 3 cc. or 5 cc. sample of the standard solution is counted in an identical manner.

5. The calculation of total blood volume (TBV) is:

$$\frac{\text{net counts (std)}}{\text{net counts (blood)}} \times 2000 = \text{blood volume}$$

6. The hematocrit may be determined by withdrawing 1 cc. of blood before the injection of the IHSA and centrifuging it in a Wintrobe tube for 30 minutes at 3000 rpm. If the blood radioactivity is counted in a graduated test tube, the blood may be centrifuged in the same tube. The result is essentially the same.

7. The plasma volume (PV) and the red cell volume (RCV) are calculated from the hematocrit. From 2 per cent to 3 per cent of the plasma is trapped in the packed red cells. A uniform correction should be made for this error. An additional correction of 6 per cent or 8 per cent may or may not be made for the difference in venous and body hematocrit. In our series, both factors were compensated for by multiplying the hematocrit by 0.915.

NORMAL STANDARDS

A review of the recent literature on blood volume studies suggests that the normal total blood volume is between 65 cc./Kg. to 80 cc./Kg. of body weight. The variation between this range is influenced by the following factors: (1) weight, including loss of weight; (2) physical habitus; (3) sex; (4) age; (5) climate; (6) method of determination; (7) correction for tapped plasma; (8) correction for difference in venous hematocrit and body hematocrit.

Weight. In normal persons there is no satisfactory reference upon which to base the predicted normal volume in a standard manner. The two commonly used means of reporting blood volume, namely, cubic centimeter per kilogram of body

TABLE I

Blood Volume in cc./Kg. in 25 Patients with an Average Weight Loss of 13.2 Kg. Calculated on Basis of Usual Weight and Present Weight

	Weight in kg.	TBV cc/kg.	RCV cc/kg.	PV cc/kg.
Usual..............................	77.1	69.7	27.0	42.7
Present............................	59.9	90.4	35.0	55.5

weight and cubic centimeter per square meter of surface area, seem equally reliable; but cc./Kg. of body weight is more generally used.

Examination of the recent literature on blood volume studies suggests that the normal total blood volume is between 65 cc./Kg. and 80 cc./Kg. with an average of 73 cc./Kg. It is difficult to set a normal standard for blood volume. Blood volume is more closely related to *lean body weight* than to any other factor, and increases per unit of body weight with increasing density. Relatively less blood is contained in fat than in muscle. It has been estimated that from 20 per cent to 25 per cent[29] of the blood is in the pulmonary circuit. In the dog, perhaps a third of the blood is contained in the abdominal viscera. At least the dog experiments by Gregersen, Cizek and Allen[20] indicate a higher volume per kilogram of viscera than of the remainder of the body.

Loss of Weight. If a patient has suffered a great weight loss, large discrepancies will appear between blood volume calculated on the basis of usual weight and blood volume calculated on the basis of present weight. Table I shows the blood volumes when calculated on the basis of present weight and on usual weight in 25 patients who had lost an average of 17.9 Kg. of weight. It is seen that if the volume is calculated on the basis of present weight, the volume is even higher than normal expectancy; but if calculated on the basis of usual weight the total blood volume and the red cell volume are less than what might be expected. All calculations of normal expected blood volume should be on the basis of usual weight before illness.

Physical Habitus. Total blood volume is relatively greater in the tall, lean, muscular person than in those inclined to obesity. Total blood volume is higher in the active than in those persons of sedentary habits. Bed rest has been found to produce a decrease of TBV of 5 or 6 per cent in two weeks, after which TBV becomes constant.[29]

Sex. Total blood volume is higher in men than in women. This may be explained by the greater amount of *lean weight* in the male.

Age. A gradual rise in the total blood volume continues up to middle age in both sexes until an average norm is reached of about 73 cc./Kg. Beyond middle age the volume per kilogram gradually declines; this is especially true in women. It may be that the actual blood volume remains the same but an increase in weight accounts for the difference in blood volume as measured in cubic centimeters per kilogram.[5, 10]

Climate. Blood volume is at its lowest at sea level and increases with high altitude.[17] It is increased somewhat in Eskimos in the winter and diminishes in

the summer.[11] A similar variation has not been reported regarding the temperate climates. Sharp reduction in body temperature may diminish blood volume. In the hypothermic dog there is a drop in TBV and RCV of about 10 per cent.[15, 28]

Method of Determination. The values for blood volume will vary somewhat according to the method used in its determination. Storaasli[30] and Aust[4] and their associates reported that plasma volumes measured by means of T-1824 dye are greater than those determined simultaneously by means of IHSA. Schultz and co-workers[27] and Brady[9] found only a relatively insignificant difference between the two methods when performed simultaneously. Determinations using red cells tagged with P^{32} give lower values for RCV and TBV[9] than does the method using cells with $Na_2Cr^{51}O_4$.[23] Chromic chloride gives results similar to IHSA and T-1824.[19]

Correction for Trapped Plasma. It is unlikely that more than 2 or 3 per cent of plasma is trapped in the packed red cells in the hematocrit.[31] It is always necessary to correct for this error if one is calculating plasma volume and red cell volume. The figure for total or whole blood volume using the technic described requires no correction for this factor.

Correction of Difference in Venous Hematocrit and Body Hematocrit. If total red cell volume is calculated directly by using cells tagged with P^{32} or $Na_2Cr^{51}O_4$ and the plasma volume is calculated separately by means of IHSA or T-1824, it will be found that the proportion of red cells to plasma found throughout the body, that is, body hematocrit, is less by 6 per cent to 8 per cent than that found by centrifuging blood withdrawn from the antecubital vein, that is, the venous hematocrit.

The venous hematocrit uniformly gives a higher percentage—6 per cent or 8 per cent—of red cell volume than actually exists throughout the body. The distribution of *extra plasma* has been investigated by Allen and Reeve[3] in dogs. They found that the hepatic and renal blood contained 60 per cent of the *extra plasma* and that the remaining 40 per cent was distributed in other tissues. They also found that the spleen contained 70 per cent more cells than the venous blood and acted as a variable reservoir for red cells. Most authors have estimated a correction factor of .910 for both trapped plasma and difference in venous and body hematocrit.

The ratio of venous hematocrit to body hematocrit is a fixed constant over a wide hematocrit range[12] and a correction for the difference is not mandatory. We have made such a correction, however, in our reports by multiplying the hematocrit by the factor .915, and we have accepted the value for TBV as 73 cc./Kg.; for RCV as 30 cc./Kg.; and for PV as 43 cc./Kg., with the corrected hematocrit as 40 per cent.

CLINICAL CORRELATION BETWEEN HEMATOCRIT-HEMOGLOBIN-RED CELL COUNT AND BLOOD VOLUME

Because heretofore most of us have not had available a routine inexpensive method of determining blood volume, we have not had an opportunity to evaluate the clinical significance of blood volume determination. Is the test reliable for
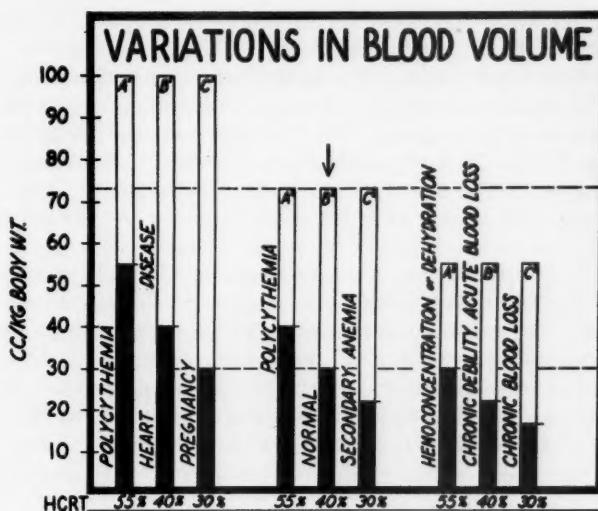
Fig. 1. Variations in blood volume

such an evaluation? We have examined approximately 500 blood volume determinations and have concluded that the blood volume determination by means of IHSA is as accurate as an evaluation made, for example, on the basis of the red cell count. An error of plus or minus 5 per cent is the normal expectation, a higher per cent of error is not at all impossible.

The second question one may ask is: "does the blood volume add any necessary information that cannot be obtained from the routine blood examination?" The red cell count and hemoglobin percentage, in addition to a clinical evaluation of the patient's status, will frequently but not always furnish adequate data for clinical purposes. It is difficult to make an informative statistical analysis of the correlation or lack of correlation between the hematocrit-hemoglobin-red cell count, on the one hand, and the blood volume on the other. It is difficult to make any reliable generalizations as to when a discrepancy may appear between these factors, but it does appear frequently. Several examples will be discussed later.

Variations in Blood Volume. There are nine possible variations in blood volume as shown in figure 1 with examples of each: A^1, B^1, C^1; A^2, B^2, C^2 and A^3, B^3, C^3. The first group is an increased total blood volume with A^1 a high hematocrit, B^1 a normal hematocrit, and C^1 a low hematocrit; the second group represents a normal total blood volume, and the third group, a low total blood volume with the same variations in hematocrit. On the whole, it may be assumed that the red cell count and hemoglobin determination will vary directly as the hematocrit varies. The ordinary hematologic examination by no means always indicates the true state of red cell mass or blood volume.

Increased Total Blood Volume (A^1). An increase in TBV with an increased RCV and hematocrit is seen in polycythemia, either primary or secondary.

Inasmuch as this finding is present in both conditions the blood volume does not help differentiate the two conditions.[6]

Increased Total Blood Volume (B^1). An increase in TBV with a normal hematocrit may be found in conditions associated with chronic oxygen deficits and fluid retention such as congestive heart disease, congenital heart disease and arteriovenous fistulas. Hyperthyroidism and overtransfusion also are associated with an increased TBV of this type. It is evident that a routine blood count and hematocrit may offer no clue as to the existence of any of the conditions mentioned.

Increased Total Blood Volume (C^1). An increase in TBV with a low hematocrit but normal red cell mass is common in pregnancy. Sometimes an increase in plasma volume with a consequent increase in TBV may simulate anemia. Plasma volume in pregnancy continues to rise to reach a maximum of 22 per cent above normal at 32 to 34 weeks of gestation.[1, 13] This period of maximum TBV seems to coincide with a period of higher incidence of cardiac failure. Berlin[7] demonstrated that in the immediate postpartum period, a decrease of 1100 cc. TBV and a return to essentially normal condition. It is interesting to note that Berlin[8] and his co-workers as well as Freis[18] did not find the usual increase in blood volume in pregnant women with eclampsia and preeclampsia.

Normal Blood Volume (A^2). A normal TBV with a high RCV is an uncommon clinical finding and is classified under polycythemia.

Normal Blood Volume (C^2). A normal TBV with a low RCV and high PV is the characteristic finding in secondary anemia. Prior to the compensatory increase in plasma volume that takes place in secondary anemia the blood count and hematocrit may be normal. As the plasma volume increases the blood count and hematocrit reflect the true degree of anemia or red cell deficit. This will be influenced somewhat if the anemia is of the microcytic type; likewise if the plasma volume increases out of proportion to the reduction in the total red cell volume, then the blood count and hematocrit will tend to exaggerate the degree of anemia. In severe chronic secondary anemia, especially in the chronic elderly debilitated patient, the blood volume may be reduced to below normal. (See Reduced Total Blood Volume B^3.)

Reduced Total Blood Volume (A^3). A reduction in TBV occurs in a fairly large variety of pathologic conditions which lend themselves particularly well to blood volume studies.

A reduced total blood volume with a normal red cell volume and increased hematocrit are found in the hemoconcentration of burns, crushing injuries, and in dehydration as seen in acute intestinal obstruction and in inadequate fluid replacement regardless of the cause. The elevated blood count, therefore, is fairly consistent with the hematocrit finding. The situation also is seen in relatively normal individuals with relative polycythemia.

Reduced Total Blood Volume (B^3). A concomitant loss of plasma volume and red cell volume is found in acute blood loss, chronic debility (chronic shock) and cardiac shock.[2, 22]

Reduced Total Blood Volume (C^2). A reduction in total blood volume and red

cell volume with a low hematocrit may follow chronic blood loss especially if hydration is poor or the plasma protein production has been exhausted. In myxedema a reduction of red cell volume and plasma volume occurs with a proportionately greater reduction of red cell volume. A reduction in total blood volume with largely a deficit of red cell volume takes place in chronic nephritis with edema.

HEART DISEASE

In a previous communication, one of us[21] reported the blood volume observations in 32 cases of congestive heart failure. Blood volume was elevated above normal in the cases studied. Total packed red cell volume was elevated more than plasma volume. In response to therapy, the plasma component was more labile and decreased more rapidly than the cellular component. Hematocrit increased with therapy. All patients observed were in decompensation and had lost an average of 13.3 per cent in weight while in the hospital. Table II is a resume of the observations in 16 patients in whom the weight was determined both before and after compensation.

TABLE II

*Mean Values for Blood Volume in 16 Cases of Congestive Heart Failure
Expressed in Percentage Deviation from Normal Values*

	TBV	RCV	PV
Calculated on basis of decompensated weight.....	+31.3	+30.5	+18.5
Calculated on basis of compensated weight.......	+50.5	+64.6	+39.9

There is some difference of opinion as to the status of the blood volume in heart disease. Reilly[24] and his co-workers report an increase in patients with right heart failure and not a great deal of change in those with left heart failure; Schreiber[26] finds an increase in volume which fell with compensation; Eisenberg[16] found an increase in patients with severe failure but little alteration in experimentally produced congestive heart failure.

A characteristic example of the alterations in blood volume in heart failure is illustrated in figure 2. A man with myocardial insufficiency due to hypertension and coronary insufficiency entered the hospital in a decompensated state. He had a secondary polycythemia with a moderate increase in the total blood volume and a marked increase in red cell volume. After seven weeks of therapy, both the total blood volume and red cell volume dropped almost to normal. He left the hospital to return in three months with some degree of heart failure and again, an elevated blood volume with mild secondary polycythemia.

ACUTE HEMORRHAGE

With the onset of severe hemorrhage, there is, to be sure, little alteration in the red cell count, hemoglobin percentage or hematocrit. Reduction takes place only after dilution by an influx of plasma fluid into the vascular compartment.
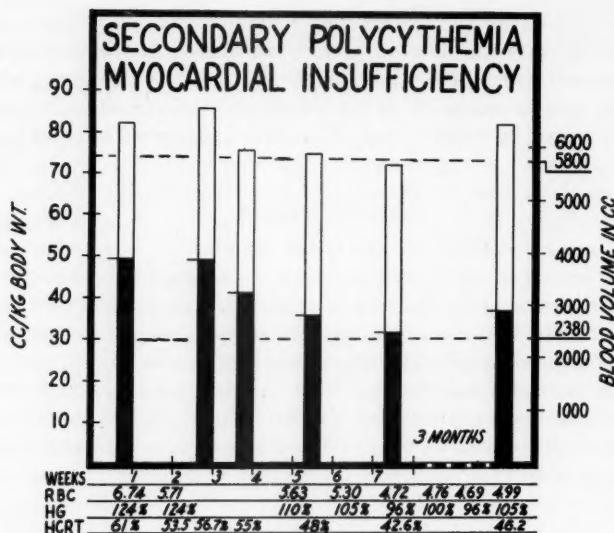
Fig. 2. Secondary polycythemia with myocardial insufficiency
TBV—Total or whole blood volume
RCV or RBCV—Red cell volume
PV—Plasma volume
RBC—Red cell count in millions
HG—Hemoglobin percentage—based on a normal average of 14.5 Gm.%
HCRT—Corrected hematocrit (venous hematocrit × 0.915)
The upper broken line represents the normal expected TBV based on 73 cc./kg. The lower one represents the normal RCV.
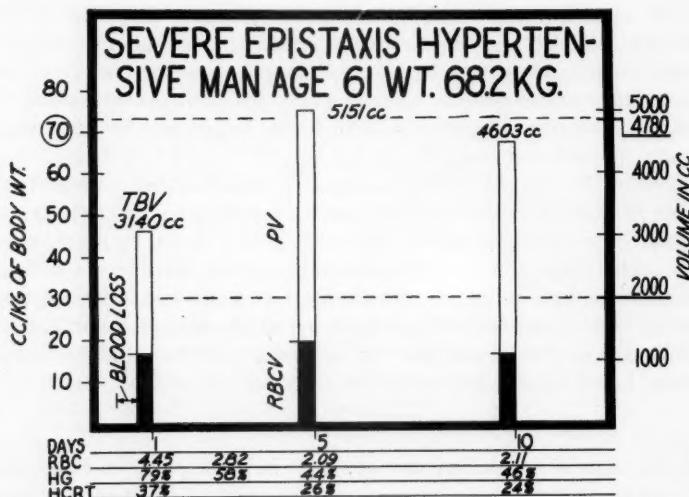
Fig. 3. Severe epistaxis in man with hypertension. Age 61. Wt. 68.2 kg.

This is exemplified in figure 3 which shows the observations in a hypertensive man with a severe epistaxis. He had been bleeding for about 12 hours before admission to the hospital. The blood count, hemoglobin percentage, and hemotocrit were normal, but a blood volume study showed a deficit of 1600 cc. The bleeding was controlled by a nasal pack. Four days later, although the blood volume had returned to a normal level, the red cell mass remained at the previous low point and the hemoglobin, hematocrit, and red cell count were reduced.

Studies of blood volume in acute hemorrhage impress one with the organism's capacity to restore plasma volume rapidly, but at the same time indicate its inability to restore red cell volume immediately. Since there is little alteration in the ordinary hematologic status in the beginning of an acute hemorrhage, the patient's condition is evaluated on the basis of his general appearance coupled with the blood pressure and pulse rate readings. An evaluation based on those factors usually will be fairly adequate, but a blood volume determination will undoubtedly give a better estimate of the blood volume deficit that should be replaced by transfusion. A day or two after the onset of the hemorrhage when the physician finds a reduced red blood count, hemoglobin and hematocrit, he then may use these determinations as criteria on which to base the need for blood transfusion. Such criteria are by no means foolproof. In the severe continuous hemorrhage, the ability of the body to restore plasma volume is sharply reduced. Blood that is transfused into the vascular compartment may not be diluted, and the red cell count and hemoglobin percentage may be normal notwithstanding volume deficit. Figure 4 shows such a situation. A woman with a severe retroperitoneal hemorrhage from a cyst of the kidney, and a low blood count, was
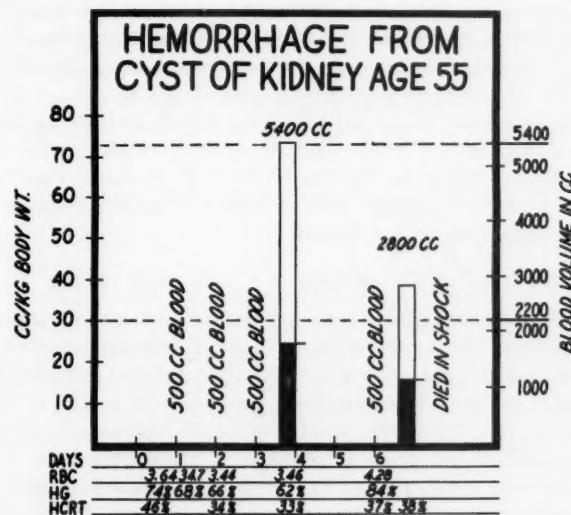
FIG. 4. Hemorrhage from cyst of kidney. Age 55
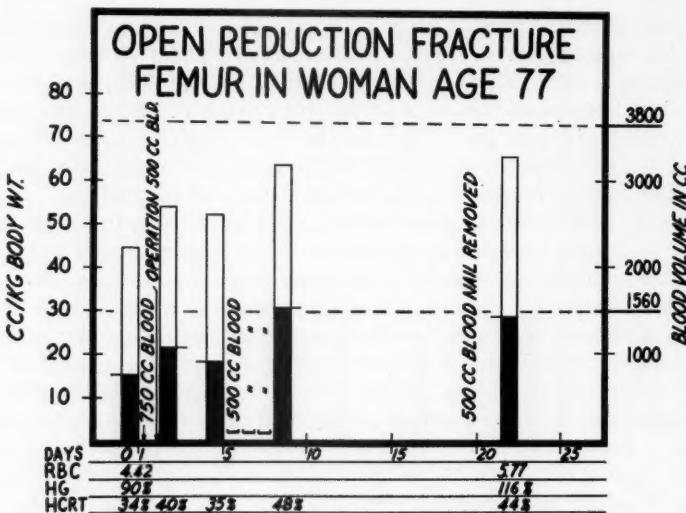
FIG. 5. Open reduction of fractures of femur. Woman. Age 77

transfused with 500 cc. of blood on three successive days. The total blood volume was raised to a normal level, the red cell volume to less than normal. Later, when the blood count was found to approximate normal figures, the indication for transfusion did not seem urgent, although it was obvious that the general condition was not good. The patient rapidly went into shock. A few hours before death, the blood volume was found to be about half normal expectancy. It is difficult to establish definite danger levels of blood volume deficits; however, as a rule, it can be assumed that when the red cell mass is only 50 per cent of normal expectancy, the situation is critical.

Red cell deficit is more important than plasma deficit. If in the presence of a red cell deficit, total volume is raised by means of an increase in plasma volume, adequate circulatory functions may be possible. If, however, there is a 50 per cent reduction in total volume as well as red cell volume, the situation is critical, and blood transfusion becomes imperative.

FALL IN BLOOD VOLUME DUE TO CLOSED INJURY

Figure 5 shows the findings in an elderly woman with a recent fracture of the femur. A preliminary blood volume showed a deficit of almost 50 per cent in both total blood volume and red cell volume notwithstanding a normal blood count. This observation led the surgeon to give a transfusion of 750 cc. of blood before operation and 500 cc. during operation. The blood volume was elevated somewhat but the condition remained unsatisfactory. Three days later, three successive daily transfusions were given of 500 cc. each. It became evident that a deficit actually existed and that the patient should have received this amount preoperatively. The drop in blood volume due to a closed fracture or crushing injury

may be excessive. Because it is accompanied by loss of large quantities of plasma as well as red cells, there will be little alteration in the red cell count or hematocrit. The patient described was elderly, and reduced blood volume is not uncommon in the aged.[5]

POSTOPERATIVE SHOCK

A man, aged 59, was subjected to a four hour gastric resection for a gastrojejunal ulcer. During the operation, he received 5 units of blood of 500 cc. each. It was estimated that he lost 1500 cc. during the operation. For the next 16 hours he remained in a state of shock. The red blood cell count was 4,880,000 per cu. mm. and the hemoglobin 14.9 Gm. per cent. Despite the normal blood findings a blood volume determination showed a total blood volume deficit of 2736 cc. and a corrected hematocrit of 31.6 per cent. The patient was given another 1500 cc. of blood and promptly recovered from the shock.

Individual examples, such as this, can be found often in any hospital where blood volume determinations have become a routine laboratory procedure.

ACUTE INTESTINAL OBSTRUCTION

A woman, aged 72, had an intestinal obstruction of 10 days duration as a result of a strangulated femoral hernia. On admission to the hospital the red blood cell count was 4,100,000 per cu. mm. and the hemoglobin was 92 per cent. Despite these normal values, total blood volume was 36 cc./Kg., the deficit being 2000 cc.; the plasma volume was 21.5 cc./Kg.; the red cell volume 14.5 cc./Kg., and the corrected hematocrit 41 per cent. At operation the bowel was found to be perforated. During operation the patient was given 750 cc. of blood and 1000 cc. of other fluids. During the operation the heart suddenly ceased to beat and could not be resuscitated by cardiac massage. Death was attributed to cardiac arrest. It is not justifiable to explain this death solely on the basis of reduced blood volume; but it certainly contributed importantly. Any patient with a 50 per cent loss of circulating blood volume, on this basis alone, is in a critical situation.

LOSS OF PLASMA VOLUME IN THE "DUMPING SYNDROME"

Walker[32] and co-workers have demonstrated a sharp loss in plasma volume during the dumping syndrome which often follows subtotal and total gastrectomy. The loss of PV appears in about 10 minutes following the ingestion of a causative meal; persists for one or two hours, and varies from 400 to 700 cc. It is accompanied by a rise of several points in the hematocrit. They attribute the symptoms in a large measure to the influx of fluid into the intestinal tract following the rapid hydrolysis of a large quantity of food, especially carbohydrates.

CHRONIC DISABILITY COMPLICATED BY SURGERY

Figure 6 represents the findings in a 66 year old obese woman with an ununited fracture of the femur which had kept her confined to bed for two years. Her total blood volume was 5600 cc. rather than the expected 9100 cc. In an obese person,
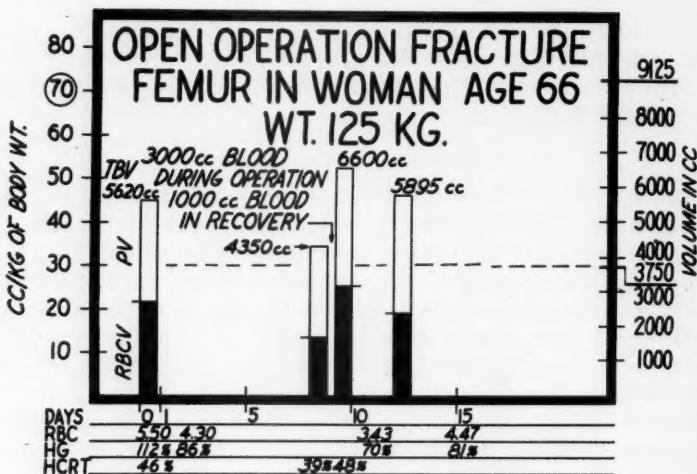
Fig. 6. Open operation in fracture of femur in woman. Age 66. Wt. 125 Kg.

the calculated blood volume is less per kilogram than in a thin person; consequently, although the calculated volume deficit in this patient may be too large, the course of events indicated such an actual deficiency.

On a clinical basis alone, during the operation 300 cc. of blood was administered. Blood loss was estimated at a far lower amount. Immediately after the operation the total circulating blood volume had dropped to 4350 cc.; the need for more blood was apparent. After the infusion of only 1000 cc. of blood, the circulating volume increased by 2350 cc., or 1350 cc. more than a mathematical calculation would permit. Some of the excess can be accounted for: The patient had undergone continuous transfusion and the exact status of the inflowing blood was not observed at the time of the blood volume determination; some of the excess may be explained by the technical errors presumably inherent in the method of determination. But this phenomenon occurs often enough to suggest that the excess is not due to technical errors. The technic does not measure volume as one would measure the contents of a vessel; rather, it measures *circulating blood volume* which may be entirely different from the actual volume volume content of the vascular tree. A patient in shock apparently has far less circulating blood volume than the actual volume, both circulating and stagnant, contained in the vessels. One transfusion of 500 cc. may have little effect in increasing the circulating blood volume; a second transfusion may be a little more effective; whereas a third transfusion of 500 cc. may increase blood volume by a larger amount than 500 cc.

The need for preoperative blood transfusion usually is based on the laboratory data of hemoglobin percentage, red cell count, hematocrit, and the blood protein. The decision is tempered by the surgeon's clinical judgment; however, as Crehan[14] has stated, "a substitution of facts for impressions can be very advantageous in

the preoperative and postoperative evaluation of the surgical patient." From our observations and those of others,[5] the erroneous impression that a patient has a satisfactory blood volume is most prevalent concerning the elderly patient, especially after a period of disability. Such patients might be said to be in *chronic shock*, as described by Clark and his associates.[13] It is mostly in this group that a normal blood count gives the surgeon false assurance. It also may be said that although the calculation of blood volume is not without error, it is by far more accurate in determining the amount of blood that a patient may require than is the judgment even of an experienced surgeon.

SUMMARY AND CONCLUSIONS

The method of blood volume determination with iodinated (I^{131}) human serum albumin has been described. The normal standards and clinical variations have been reviewed. Examples of the useful applicability of blood volume findings in hospital practice have been described.

In surgical practice the factual knowledge of blood volume status is as valuable or perhaps more valuable than the red cell count, hemoglobin determination, and hematocrit, and gives information that cannot be obtained by these means.

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TUMORS OF THE COLON

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In March 1931, Dr. John F. Erdmann, of New York City, read a paper in this city, before this Congress, on the same subject that has been assigned to me. Needless to say, I esteem it a great honor to be chosen by this distinguished assembly to follow in the foot-steps of such a great clinician. I have read Dr. Erdmann's paper carefully, and have come to two conclusions that might have been surmised in advance. The first of these is that in matters dealing with clinical observation based upon a rich personal experience, there is little to add to or change in the comments of Dr. Erdmann in making a present day resurvey of the subject. The second is, that the additions that are necessary have arisen because of new knowledge, new tools and new technics that have developed since the date of the original paper.

To elaborate upon this brief statement, one may say that Dr. Erdmann, based on an accumulation of cases that was large for those days, gives an analysis of the distribution of colon cancer in regard to age, sex, and anatomic location that is almost exactly the experience of the present writer, and of that recorded in current publications. The one item that might be questioned is the statement that rectal cancer is markedly more frequent in females. My own experience fails to show any significant sex preponderance. The clinical descriptions of the signs and symptoms of the disease are full and graphic—equal to anything in today's literature. In fact, Dr. Erdmann records one sign that now is not widely recognized and that may profitably be repeated in his own words. He says, in speaking of growths of the distal half of the colon, producing partial or complete obstructions, "I have called attention for years to a metallic tinkle heard with the ear over the cecal region when the opposite side is pushed sharply toward the median line. The sound is due to a collection of fluid contents in the cecum with gas above it. I consider this an infallible sign of obstruction complete or partial."

There is the same distinction between the characteristics of lesions on the right side from those on the left that now are recognized. There is the same emphasis on marked secondary anemia as a possible indication of obscure right colon cancer. Attention is called to the greater likelihood of obstructions developing in growths on the left side, and a discussion of the reaons for this, namely, the increased consistency of the fecal contents and the decreased lumen of the intestine on the left side. There is insistence on the importance of roentgenologic examination of the bowel in making a diagnosis, and mention of the fact that at times such an examination may give trouble by the formation of barium impactions.

It is appropriate here to point out one of the technical advances, made since Dr. Erdmann wrote, that has added much to our diagnostic resources. There formerly was frequent difficulty in reading colon roentgenologic studies caused by the overlying of loops of colon on one another. The development of improved fluoroscopy, of lateral and oblique positioning, and especially of air-contrast studies by inflation following barium enemas, have given the diagnosis of colon conditions an accuracy that did not exist 25 years ago.

The difference of *now* and *then* is more evident in the field of treatment. Dr. Erdmann uses terms in regard to the preoperative preparation of patients that have a surprisingly up-to-date sound. For instance, he says: "Thorough intestinal cleansing should be done, when possible by cathartics, intestinal antiseptics, and colonic irrigations." But we know that decompresssion by long tube suction is much better than cathartics. We also have, in the sulfa drugs and the antibiotics, means of intestinal disinfection far superior to the very doubtful ones then existing.

It is in the specific methods of treatment that one notes the greatest change resulting from the accumulation of 25 years of added experience and gradually improved technics. Dr. Erdmann clearly recognized the basic principles that applied to the choice of operative methods in various conditions. He stressed the necessity of wide removal of the disease for attempts at radical cure; the wisdom of local resections as a palliative operation when cure is out of the question because of distant metastases, and the danger of extensive operation in the acutely obstructed intestine. He was writing, however, before the advances just mentioned had made the preliminary relief of obstruction in many patients so satisfactory that the stress he laid on preliminary colostomy now is often believed to be unnecessary. One of the notable differences in present day surgery from that current when Dr. Erdmann was a foremost surgical teacher is the widespread tendency toward single stage operations, even of great magnitude, rather than the multiple stage attacks then thought to be necessary for safety.

It remains true, however, that where relief of obstruction cannot be secured promptly by medical measures, a preliminary colostomy is necessary before the definitive attack on the disease itself is undertaken. The present writer prefers a transverse colostomy when the growth is distal to the splenic flexure, rather than the cecostomy advocated by Dr. Erdmann, and he believes this is currently the general opinion of surgeons.

This brings us to what is perhaps the greatest practical difference in the actual surgical procedures advocated by Erdmann and those now generally approved. Dr. Erdmann specifically stated that he preferred the perineal resection for anal, rectal, and low rectosigmoid growths. He said, "The extensive operation of Miles I cannot, at present, feel is called for, particularly after closely analyzing his immediate and remote mortality." To one who personally remembers the mortality rate in the early experience with the abdominoperineal principle advocated by Miles, this reaction is not surprising. Again, however, the wide use of blood transfusions, the correction of the blood-chemistry and fluid balance by preoperative measures and the use of shock-combatting steps during operation,

have put an entirely different face on the matter of mortality rate. These and other refinements of technic have reduced the mortality rate of the present Miles operation, far below that of the perineal method of Dr. Erdmann's day. With the winning of this greatly enhanced safety, the obviously sound principles of wide anatomic removal of tissues subject to invasion by disease has become generally accepted as practical and now is predominantly utilized. In fact in the last few years the original Miles operation has been extended by a number of surgeons to include the ligation of the inferior mesenteric artery at its source and the removal of most or all of the descending colon, in addition to the scope of removal described by Miles himself.

Dr. Erdmann recognized and emphasized the occurrence of multiple tumors of the colon either simultaneously, or successively. He did not mention, however, the most interesting and most serious form of multiple lesions, namely, true multiple adenopolyposis of the colon. This interesting condition often is of familial incidence. Its chief importance lies in the marked tendency of these polyps which are true adenomas, to become malignant. Many of the victims of this disease die of cancer of the bowel at remarkably early ages. Increasing recognition and study indicate that in this condition the mucosa of the entire colon, or at least large segments of it, has an abnormal tendency to grow polyps, sometimes literally hundreds of them, and that these polyps, as has been said before, tend to become malignant. The consequence of this state of affairs is that the safety of the patient demands the removal of the entire large bowel. Any less complete eradication of the tumor-forming mucosa does not eliminate the threat of future cancer development. At the time of Dr. Erdmann's paper such radical surgery was not contemplated, and the disease we are considering here was not sufficiently understood to be discussed by him at all. Now total colectomy is employed, not only for multiple adenopolyposis but more frequently for the nonmalignant condition of advanced chronic ulcerative colitis,—although this too may at times terminate in areas of malignant degeneration. Here again we note the modern tendency toward employing much more radical surgery.

When one thinks back to the position of Dr. Erdmann in his prime, of his courage, initiative and sound judgment, one can conclude only that were he writing this paper today he would rejoice in the advances that make possible the differences noted between 25 years ago and today. We hope that whoever writes the third paper 25 years hence may have much greater advances to record.

SYMPTOMATIC LESIONS MASKING CANCER
OF THE LARGE BOWEL

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The tendency of cancer to cloak itself in the symptoms of coexisting disease, usually of a much less serious nature, often is encountered. Perhaps nowhere in the body does this occur more frequently than in the colon and rectum—a fortunate circumstance, for this portion of the gastro-intestinal tract is accessible to diagnostic methods which are fairly accurate in differentiation.

It is the purpose of this paper to review briefly the chief causes of confusion in this area and to present 4 illustrative cases, reaffirming the importance of keeping malignant disease in mind when treating cancer-age patients for any lower bowel complaint, and stressing the necessity of using all diagnostic aids available, repeatedly if results are equivocal, before assuming that cancer is not present. It is hoped that this discussion will contribute toward consistency in the early diagnosis of large bowel cancer.

DISEASES MASKING CANCER

Diseases most commonly masking cancer of the large bowel are appendicitis, functional disorders, diverticulitis, bleeding polyps, amebiasis, chronic ulcerative colitis and hemorrhoids (table I). Differential diagnosis of these lesions will not be considered, but rather the early diagnosis of obscured coexisting cancer.

Appendicitis. Continued right lower quadrant discomfort in a patient over 35 years of age should raise the question of cancer, even though there is a history of protracted medical treatment for what was thought to be chronic recurrent appendicitis. This is especially true when there is weight loss or anemia. Appendiceal abscess does not often mask cancer although it occasionally is involved in an erroneous diagnosis.

Functional Disorders. Confirmed neurotic patients all eventually succumb to a fatal illness, in some instances cancer. So-called *mucous colitis* or *irritable colon*, present for years, may conceal developing malignant new-growth, and functional disturbances occurring during the menopause may blunt the physician's alertness to the possible presence of cancer.

Diverticulitis. The occasional close resemblance of sigmoid diverticulitis to carcinoma is well known. It also should be remembered that these diseases may occur simultaneously. In a patient having diverticulitis, after subsidence of fever, tenderness and leukocyte count, persistence of obstructive symptoms or localized roentgenographic filling irregularity warrant resection of the segment.

Chronic Ulcerative Colitis. It is not unusual for carcinoma of the colon to originate during the course of ulcerative colitis, and such a carcinoma usually is highly malignant and invasive. Its onset in most instances follows that of ulcerative colitis by several years, but it may originate at any time. Obviously it

TABLE I

Lesions Most Commonly Masking Cancer of Large Bowel

Appendicitis			
Functional disorders			
Diverticulitis		Lt. Col.	Rt. Col.
Chr. Ulcerative Colitis	Rect.		
Polyposis			
Amebiasis			
Hemorrhoids			

may be very difficult, and in some patients it may be impossible to detect a malignant neoplasm developing in such a colon. In patients with chronic ulcerative colitis developing polyps or pseudo-polyps, as well as in those with disease of long duration involving most of the colon with poor response to medical management, it appears that colectomy and ileostomy should be done. After resection, previously undetected carcinoma will be found in some such colon specimens.

Polyps. Although bleeding polyps may on occasion obscure the diagnosis of an independent cancer, the greater danger is that they themselves might become malignant. Most surgeons agree that every polyp should be removed and its base studied microscopically. Deddish and Fairweather[1] have described a useful method for inspecting the mucosal surface of the entire colon for small polyps at laparotomy, utilizing a few advantageously placed taenial incisions and a sigmoidoscope.

Amebiasis. This disease is of especial importance to physicians practicing in the Mississippi Valley, as it has been variously estimated that between 6 per cent and 18 per cent of the population of this area are affected. Mayfield,[3] in 1953, reported 75 cases diagnosed and patients treated by him in a 12 month period in a small Mississippi comunity of 10,000 in a 10 mile radius. Rudner[4], in 1953, reported a significant number of Mississippi Valley surgeons who stated that coexisting amebiasis is one of the major causes of delay in diagnosing carcinoma of the colon. Amebiasis should be treated when detected, but in patients over 35 years of age investigation should not slacken until a complete study of the large bowel has been made. This should be done repeatedly if bleeding persists after treatment for amebiasis. Once a double diagnosis of cancer and amebiasis has been made extreme caution must be exercised, because colon surgery in the presence of amebiasis might prove disastrous, with peritonitis and fistula resulting. Goldberg and Stegman[2] suggest that the occasional failure of suture lines following colon resection and anastomosis might well be due to unsuspected Endameba histolytica in the bowel. Certainly it would seem a wise precaution to obtain stool studies on all candidates for elective colon surgery, whether or not they have had symptoms suggestive of amebiasis.

Hemorrhoids. In any patient with rectal bleeding the known presence of hemorrhoids imparts a false sense of security. A sigmoidoscope should be passed before advising hemorrhoidectomy in every patient, and this should be supplemented with barium enema studies in all patients in whom the rectosigmoid

flexure cannot be negotiated with the sigmoidoscope, or when this examination shows normal bowel and there remains some doubt as to whether the hemorrhoids are the sole source of bleeding.

REPORT OF CASES

The following 4 cases of masked large bowel cancer from a modest surgical practice in a small city were encountered since August 1952, a rate of almost 2 per year. The first case does not fall into the preceding outline, but is appropriate to the purpose of this discussion.

Case 1. A 69 year old white man was admitted to the hospital on Aug. 24. 1952 with an acute large bowel obstruction and impending rupture of the cecum. He gave a history of increasing constipation and recurrent rectal bleeding for approximately one year. During the last six months he had very frequent small stools with much bloody mucus, accompanied by weight loss from 150 to 117 pounds. His last stool was passed four days prior to admission to the hospital and the picture of acute obstruction developed rapidly thereafter. The referring physician reported that several weeks before development of obstructive symptoms he had made a sigmoidoscopic examination and found only a small polyp at about 13 cm. above the anus. Upon admission to the hospital emergency cecostomy was done, and the ensuing 10 days were devoted to restoration of nutrition, blood volume and electrolytes, and preparation of the bowel for resection. During this period a barium enema roentgenologic study showed a completely obstructing lesion high in the sigmoid and plans were made for definitive surgery. The day before operation the referring physician's mention of a small polyp was remembered, prompting a repeat sigmoidoscopic examination and biopsy of the polyp. The pathologist reported adenocarcinoma, grade I; consequently, in carrying out anterior resection the following day the lower level of resection was placed distal to the polyp, which was so small that it could not be positively identified by palpation through the bowel wall (fig. 1). This malignant polyp was located so far distal to the obstructing carcinoma that it would surely have been left had its presence not been known, and the later clinically apparent cancer might well have been labeled *recurrence*. At the present time there has been no evidence of recurrence (March 1955).

Case 2. A 46 year old white housewife was seen April 14, 1953 complaining of moderate dyspepsia of many years duration with occasional nausea and vomiting. There had been no recent aggravation of this complaint. Her menses had become irregular, with increased flow, and she showed evidence of moderate anxiety with functional vasomotor phenomena common to the menopause. She thought she had lost about 15 pounds over a period of four years, but she weighed 122 pounds on examination and appeared well nourished. In the course of examination a fusiform, mobile mass was palpated in the right upper quadrant of the abdomen. She then was questioned carefully concerning her bowel habits and character of stools, but no history of alteration of either could be elicited, except for softer consistency of stools during the past eight months. She had never seen evidence of bleeding in her stool. Barium enema studies showed a large tumor mass involving the colon at the hepatic flexure. Her erythrocyte count at this time was 4,010,000 red cells per cu. mm. and her hemoglobin 11.4 Gm. On April 24 resection of the ascending and transverse colon was done and a large ulcerating lesion was found (fig. 2). The pathologist reported adenocarcinoma, grade IV, with metastases to a cluster of mesenteric nodes adjacent to the bowel. The nodes removed from the root of the mesentery showed no metastasis. The liver appeared to be normal. She has not yet shown evidence of recurrence (March 1955).

Development of softer consistency of stools, in view of long-standing functional dyspepsia and approaching menopause, hardly seems worthy of alarm, yet apparently it was the *only symptom* referable to the advanced cancer in this woman's colon.

Case 3. A 57 year old white housewife was first seen in the office on May 18, 1953, one month after the referring physician first saw her because of diarrhea with bloody stools.

69 yr W ♂ 8/24/52
Progr. Constipation 1 yr.
Rectal bleeding 1 yr.
wt. loss (33 lb.)
Complete Obstruction 4 da.
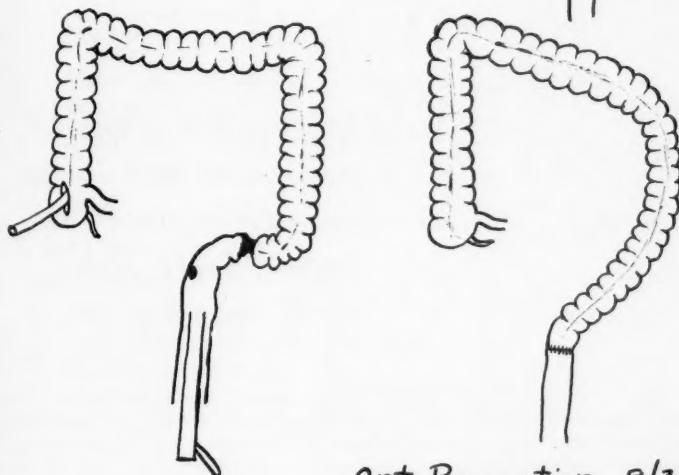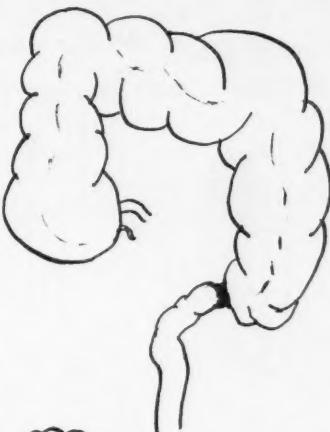
Ant. Resection 9/3/52
(Adenoca of Sigmoid, Grade II)
("   "   Polyp, Grade I)
L. + w. 2½ yrs.

Case no. 1.

Fig. 1

He reported that sigmoidoscopic examination showed no abnormalities except internal hemorrhoids; however, a smear of sigmoid contents was found to contain numerous trophozoites of E. histolytica. Barium enema studies made at the same time showed no filling abnormality of the colon. He began amebacidal treatment with Neobacin, and all symptoms soon subsided. Referral was made one month later when there was recurrence of diarrhea and bleeding, and the stool again was found to contain E. histolytica trophozoites. Diodoquin was prescribed, and the symptoms promptly began subsiding. On June 22 she reported occasional rectal bleeding. Stool examination showed no E. histolytica trophozo-

46 yr W ♀   4/14/53
Dyspepsia many yrs.
Metromenorrhagia 4 mo.
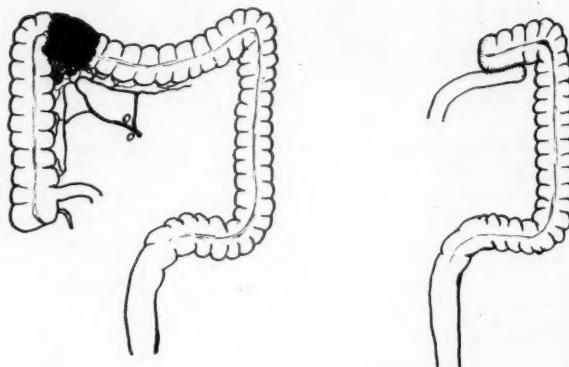
Case no. 2.

Rt. Colectomy 4/24/53
(Adenoca Gr. IV, c̄ metas-
tasis to adjacent mes-
enteric lymph nodes)
L. + W. 1½ yrs.

Fig. 2

ites or cyst forms. On June 27 sigmoidoscopic examination showed normal mucosa, but a bolus of bloody mucus was encountered at about 15 cm. from the anus. This material was examined microscopically but no trophozoites or cyst forms were found. On June 28 barium enema studies first were reported normal then, on re-examination by a consulting radiologist, as showing evidence of a possible intrinsic lesion in sigmoid flexure. On August 22 a course of emetine therapy was begun, and after completion additional barium studies were made. These were reported as showing a filling defect in the sigmoid flexure. Anterior resection and anastomosis was done on September 6 and the pathologist reported adenocarcinoma, grade III, with reactive hyperplasia of adjacent modes (fig. 3). This patient had a stormy convalescence beginning on the second postoperative day with high fever and progressive abdominal distention. Complete obstruction developed at the site of anastomosis and three days of Miller-Abbot suction, antibiotics and supportive therapy were necessary before flatus began passing through the anastomosis; after this, recovery was rapid. In retrospect it appears possible that these complications were due to E. histolytica in the bowel, in spite of the vigorous preoperative amebacidal therapy and subsequent stools free of amebas. This patient was fortunate not to have had anastomosis suture line failure. Since surgery there has been no evidence of recurrence of either amebiasis or carcinoma (March 1955).

    Case 4. A 73 year old white housewife stated that she had been under a physician's care

57 yr. W♀ 5/18/53 ,
Diarrhea c̄ bleeding 4 mo.
  4/18/53 Saw ref. Phys.
    Sigmoido: Internal Hem.
    Stool: E. Histolytica troph.
    B.E.: "Normal bowel"
    R̡: Neobacin (subsidence of sympt.)
  5/8/53 Recurrence
  5/20/53 Stool: E. Histolytica
    Rx: Diodoquin
  6/22/53 Occas: rectal bleeding
    Stool: Normal (No troph. or cysts)
  6/27/53 Sigmoido.: Normal up to
    15 cm., encountered bolus of
    bloody mucous (Neg. for E.H.)
  6/28/53 B.E.: "Poss. intrinsic
    lesion in Sigmoid flexure"
  7/22/53 Emetine therapy begun
  7/31/53 B.E.: "Filling defect in
    sigmoid flexure."
  8/6/53 Anterior resection
    (Adenoca Grade III, c̄
    reactive hyperplasia of
    Adjacent nodes).
      L. + W. 15 mo.

Case no. 3.

Fig. 3

for three years because of arterial hypertension and various gastrointestinal tract complaints, including bleeding hemorrhoids. The physician's case record transcript indicated that he saw her at frequent intervals during period, and that on Aug. 22, 1952 he did a rectal examination and barium enema, noting the presence of internal hemorrhoids and sigmoid diverticulosis.

On Nov. 30, 1953 she was seen in the office because of the same complaints. During physical examination the previously reported internal hemorrhoids were noted, and in addition

73 yr W ♀ 10/30/53
Flatulence ⎫
Hemorrhoids ⎬ 3 yrs.
 (bleeding) ⎭
Px : Polypoid lesion c̄
 broad base @ 4 cm.
Biopsy : Adenoca Gr. I
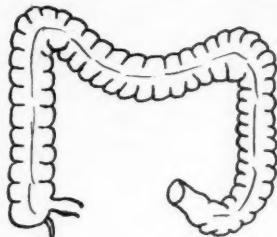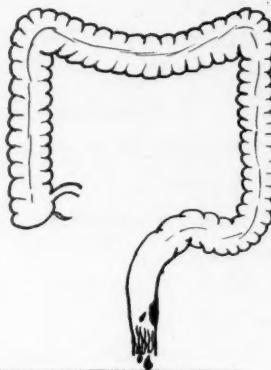
Comb. Abd. Per. Res. 11/20/53
L. + W. I yr.

Case no. 4.

Fig. 4

there was found a polypoid growth with a broad base measuring approximately 2.5 by 3.5 cm. on the posterior wall of the rectum just above and contiguous with the upper extremity of a hemorrhoidal mass (fig. 4). Biopsy disclosed adenocarcinoma, grade I. Combined abdominoperineal resection of the rectum was done on November 20, and she remains well at this writing (March 1955).

SUMMARY AND CONCLUSIONS

A brief review of lesions most commonly masking cancer of the large bowel has been presented and 4 illustrative cases discussed. While these cases do not represent a large experience they may serve to remind that masking of large bowel cancer can and probably does occur in everyone's practice. They also emphasize certain points of interest: 1. Advanced, recognizable cancer in one part of the large bowel does not preclude the possibility of coexisting asymptomatic cancer in another part. 2. Large bowel cancer does not necessarily produce any recognizable symptoms, even though it may be advanced. This is especially true in the right colon. 3. Amebiasis, occurring with cancer, may cause great delay in diagnosis of the cancer, even when it is suspected and every effort is made to

reveal it. Amebiasis is one of the most insidious maskers of colon cancer, and, in the Mississippi Valley, one of the most common. 4. Routinely before elective colon surgery the stool should be studied for latent amebiasis in order to avoid serious postoperative complications. 5. Hemorrhoids still are responsible for delay in diagnosing cancer, both of the rectum and of other portions of the large bowel. 6. In every patient past 35 years of age with any large bowel complaint the possibility of cancer should be entertained, and steps taken to rule it out. These steps should include both proctosigmoidoscopic examination and barium enema studies, regardless of the apparent insignificance of the symptoms. Adoption of this policy, while producing many findings of *normal bowel*, also will bring to early operation many patients with large bowel cancer masquerading with symptoms of other coexisting disease at a time when the cancer is curable.

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DIAGNOSIS AND MANAGEMENT OF ACUTE ABDOMINAL CONDITIONS

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At the first meeting of the Southeastern Surgical Congress 25 years ago my distinguished father, John Blair Deaver, outlined existing concepts of the diagnosis and management of acute abdominal conditions. Your Society has invited me to represent him at this meeting today and to *"bring his paper up to date"*. This is an assignment I am very happy to accept and will undertake it with true humility.

Probably no diagnostic problem is more challenging to the physician than the acute abdomen, and despite medical advances in many fields during the intervening quarter century it is interesting to note that the basic tenets in evaluating this problem have changed very little. John B. Deaver stated in 1930 that "in no emergency is clinical acumen a more valuable asset", and his statement holds true today. Despite a profusion of laboratory and radiologic aids, the most vital information in diagnosis of the acute abdomen continues to be gleaned from a meticulous history and physical examination, and an astute interpretation of the findings. I would add that the earlier the acute abdomen is seen, the more confident one can be of the diagnosis; and the earlier suitable treatment is instituted, the more favorable will be the prognosis. Various laboratory data and roentgenologic studies certainly are helpful, but it should never be forgotten that they are merely adjuncts.

Acute appendicitis. The most common surgical lesion causing the acute abdomen is, of course, appendicitis and any patient presenting abdominal pain, tenderness and rigidity in whom the diagnosis seems obscure, appendicitis should be suggested as the most likely pathologic process. Next in order of frequency are intestinal obstruction, cholecystitis, perforated peptic ulcer, acute pelvic pathology, diverticulitis, pancreatitis, mesenteric thrombosis and torsion of the great omentum.

The typical case of appendicitis is relatively easy to diagnose. The atypical case may present so many difficulties that the diagnosis cannot be made except by exclusion. There is no laboratory study or roentgenologic examination which can make the diagnosis of acute appendicitis. However, in the differential diagnosis of such conditions as pneumonia and ureteral calculus, laboratory aids can be of considerable value, provided one never depends upon them *alone*, but *correlates them* with the history and physical findings. In many cases of acute appendicitis and acute perforative appendicitis, for example, the blood count is not elevated. Nor does the finding of red blood cells in the urine rule out appendicitis. On the contrary, this is common when the acute inflamed appendix lies over the ureter.

I would list in the order of their importance the following procedures in the examination for appendicitis: (1) palpation of the abdomen; (2) rectal examination; (3) a careful history; (4) laboratory and roentgenographic findings, such as blood count, urinalysis and flat roentgenogram of the abdomen. In the differential diagnosis of acute appendicitis and ureteral calculi or gravel, a flat roentgenogram of the abdomen, an intravenous urogram, and at times even cystoscopy with ureteral catheterization may be indicated. In a certain percentage of patients, even with a careful history and physical examination, a differentiation between appendiceal involvement and acute pelvic conditions, such as twisted tube or ovary, ruptured tubal pregnancy, and the like cannot be made clinically. Under these circumstances, one has no choice but to establish the diagnosis by laparotomy.

Prompt appendectomy is the treatment of choice for all cases of appendicitis except those late cases in which distention is greater than rigidity. In these, intestinal intubation, parenteral fluids and antibiotic therapy may bring about localization with abscess formation or complete resolution of the infection.

Acute mesenteric adenitis, a condition generally limited to the younger age group, I will mention only to say that I know of no clinical or laboratory test which will aid in differentiating this condition from acute appendicitis. In my experience, it seems that when I make a preoperative diagnosis of acute mesenteric adenitis, at operation we find an acute appendicitis, and vice versa.

Intestinal obstruction probably is the second most commonly encountered cause for the acute abdomen. Acute intestinal obstruction, especially of the small intestine, is a condition which demands immediate surgical treatment. The operative mortality rate from this condition is too high, and in the large majority of patients it is due to the fact that operation is not done early enough.

When taking the history, it is important to remember that the bowel distal to the site of the obstruction is normal and acts in a normal fashion. Hence, early in the obstruction there often will be passage of gas and even intestinal contents. Thus, the history of passing gas or fecal matter does not necessarily militate against the diagnosis of obstruction.

In discussing intestinal obstruction from any angle, diagnostically or therapeutically, I think we must divide the subject into obstruction of the large intestine and obstruction of the small intestine. With the exception of volvulus and intussusception, obstruction of the large intestine is far less serious than that of the small intestine. The symptoms are less pronounced and the urgency for immediate operation is decidedly less. Acute obstruction of the small intestine demands prompt surgical intervention, or else the mortality rate will be prohibitive.

Physical findings of distention, hyperperistalsis and vomiting are indicative of obstruction, and scout films of the abdomen are of great diagnostic value. But the most significant clue to diagnosis of this condition is the presence of pain—severe, cramp-like pain occurring at regular intervals. This symptom alone, all other findings being negative, I believe is enough to warrant early surgical intervention.

Scout roentgenograms of the abdomen, when properly interpreted, can yield

information of as much, or more, aid in the diagnosis of obstruction than any single clinical finding with the exception of pain. These films should be taken with the patient in the erect and lateral positions and before any intestinal decompression is undertaken or any enemas are given, or else the introduction of air by these means may confuse the roentgenographic findings.

Quoting from Frimann-Dahl's book, "ROENTGEN EXAMINATION IN ACUTE ABDOMINAL DISEASES", he states: "In our experience with the cases showing negative or slight roentgen findings the obstruction was due to complete strangulation. This leads to the serious problem of difficulties in which we may conclude: The most dangerous cases of obstruction are sometimes the most difficult to diagnose." The explanation of negative findings in these cases of complete strangulation is that the involved portion of the intestine immediately fills with fluid, excluding any air from the involved loop, and thus presenting an apparently normal roentgenographic picture.

In obstruction of the small intestine, early operation is imperative and is aimed at relieving the obstruction. If resection is necessary, primary anastomosis only is done and exteriorization is never attempted.

In obstruction of the large intestine, volvulus and intussusception demand immediate surgery. If resection is indicated, primary anastomosis is done, with or without proximal decompression, and exteriorization may be done in certain selected cases. If the obstruction is due to tumor, attempts should be made to relieve the obstruction by nonsurgical means, such as intubation and the like. If this fails, some form of proximal decompression operation should be done as a first-stage procedure.

Perforated gastric and duodenal ulcer: Perforation of a peptic ulcer into the peritoneal cavity is a catastrophe which occurs with dramatic suddenness. Fortunately, it is one of the most easily diagnosed of the acute abdominal emergencies. The abdominal wall is boardlike and tender. Respiration is shallow and of the costal type. Varying degrees of shock may or may not be present, and the pulse and temperature are of little help in diagnosis. The blood count usually is elevated, although this is not always so.

In this condition, the greatest help in substantiating and at times in making the diagnosis is afforded by roentgenograms of the abdomen, i.e., scout roentgenograms. When the roentgenograms are properly taken their interpretation can be diagnostic in an essentially high percentage of cases—95 or above. The position of the patient while a roentgenogram is being made is extremely important. Roentgenograms should be taken in the erect and in the left recumbent positions, and for the latter views the patient should have assumed the position for at least 5 or 10 minutes before the exposure is made.

Early operation, with simple closure, is the current procedure of choice. Subtotal gastrectomy, a procedure in favor with European surgeons for many years, is being done with increasing frequency in this country. However, we believe that subtotal gastrectomy should not be considered unless the following criteria are met: (1) the patient is in a young age group; (2) signs of bacterial peritonitis have not appeared; (3) there are no signs of malnutrition; (4) there

is no distention of the small intestine (not ABDOMEN); and (5) an adequate supply of blood is available and a transfusion of whole blood is started when the operation is begun. Intratracheal inhalation anesthesia is our choice.

The so-called conservative or nonoperative treatment, consisting of gastric decompression, parenteral fluids, antibiotics and the like should be resorted to only when adequate facilities for major surgery are not available, or under other exceptional circumstances.

Acute pancreatitis is certainly one of the causes of the acute abdomen, but I do not believe that it falls, per se, into the category of a surgical emergency, and we do not operate upon acute pancreatitis unless we cannot make a diagnosis without doing so. The physical findings are somewhat similar to those of perforated ulcer. The tenderness and rigidity are less pronounced and tend to be confined to the upper abdomen and epigastrium. Radiation of pain to the middle of the back is common, and varying degrees of shock is the rule rather than the exception. Elevation of the serum amylase is a very reliable laboratory finding and often clinches the diagnosis. However, although this test is very accurate, it must be done early in the disease, i.e., within 24 to 48 hours from the onset, to be valid.

Acute cholecystitis usually does not present any great diagnostic difficulties, and we believe that it rarely calls for immediate surgical intervention. In this condition the tenderness, rigidity and pain are more marked in the right upper abdominal quadrant. There generally is a previous history of biliary tract disease. Very often there is radiation of pain through to the back in the right scapular area.

Laboratory studies, such as serum bilirubin and van den Bergh tests, and roentgenograms seldom are helpful in the diagnosis. Even if stones are shown on the roentgenogram, this finding may be incidental and does not rule out pancreatitis, peptic ulcer, mesenteric thrombosis or other acute abdominal conditions.

We prefer not to operate upon the acute gallbladder immediately but to observe the clinical picture under conservative measures for from 24 to 48 hours. If there has been no improvement at that time, we operate. Cholecystectomy is the procedure of choice and can be done in the majority of cases. However, if the patient is a poor risk or the technical difficulties are great due to massive infection or edema which makes anatomic identification precarious, one should not feel chagrined to content himself with a simple cholecystostomy and defer definitive surgery to a more opportune time.

We believe that the cases of acute cholecystitis which are most dangerous and which may most often call for immediate operation are those occurring in the older age group. With the increase in geriatric surgery, it has been our experience that in this group the disease is less apt to subside and perforation is more common. In this group temperature and blood count are more apt to be normal than elevated.

Mesenteric thrombosis presents a nonmechanical type of intestinal obstruction. Usually the patient is extremely ill and shock is an early symptom. Roentgeno-

logic examination in this condition shows merely an adynamic ileus and is not diagnostic. Severe mesenteric thrombosis causes symptoms very similar to those of dissecting aneurysm, and in the absence of a typical history I know of no way of differentiating the two conditions. Clinical interpretation of the abdomen plus careful examination of the heart and cardiovascular system is of prime importance. The electrocardiogram may be the most helpful laboratory aid in establishing the diagnosis. Treatment consists of surgical resection of the involved area. This may in some cases amount to a massive resection of the affected portion of the intestinal tract.

Adominal trauma also can be included among the causes of acute abdomen. I will confine my remarks on this subject to nonpenetrating injuries of the abdomen. Injury to solid viscera causes hemorrhage. Injury to hollow viscera usually causes peritonitis. Both types of lesions are accompanied by shock. If the symptoms of shock do not respond to treatment within three or four hours, hemorrhage or peritonitis should be suspected. Circulating blood volume studies and repeated blood counts and hematocrits are distinct laboratory aids in these cases. Immediate scout roentgenograms of the abdomen also should be taken. The presence of free air in the peritoneal cavity is very strong evidence of perforation of a hollow viscus, and such a finding calls for immediate operation.

CONCLUSION

In conclusion, I wish to re-emphasize that the most important part in the diagnosis of any acute abdomen is a meticulous physical examination in conjunction with a careful and thorough history. Laboratory technics, while of undeniable assistance, should never be regarded as more than adjunctive in nature, and should be evaluated only in the light of the complete history and physical findings.

A general axiom which might be well worth remembering in dealing with any acute abdominal emergency is this: Severe abdominal pain of six hours' duration in a previously well patient usually indicates the need for surgical intervention.

THE PRESENT STATUS OF GALLBLADDER SURGERY

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Twenty-three years ago, at the 1932 meeting of this Congress, I gave an address entitled, "The Present Status of Surgery of the Gallbladder." I was requested to speak on the same subject on this anniversary, presumably because it is an appropriate occasion for looking backward to see what has been accomplished in surgery, particularly of the gallbladder, during the years since the organization of our group.

In this brief life span of ours we consider a quarter century a long time, as indeed it is. On first thought, therefore, we may be inclined to have a dim view of the status of all surgery 25 years ago. As a matter of fact, at the time this organization was founded, surgery in general was almost as far advanced as it is today. With respect to disease of the gallbladder, the etiologic factors were fairly well understood, diagnostic methods were highly developed, principles of operative technic were clearly established, mortality following cholecystectomy was, on the whole, 5 per cent or less, and the outcome was successful in approximately 85 per cent of the cases. Nevertheless, in considering the entire gallbladder problem from the current viewpoint, a noteworthy progress may be observed in most of these respects and in others as well.

A quarter century ago the etiologic factors in gallbladder disease were believed, as now, to be a disturbance of cholesterol metabolism, chemical imbalance, reflux of pancreatic secretions into the biliary system, and hormonal elements which, acting upon both the autonomic and central nervous systems, regulate the contraction and evacuation of the gallbladder. Probably the chief difference from current opinion as to the etiology concerns the influence of infection. Whereas formerly infection was regarded as a prominent factor, in the majority of cases it now is believed to play a secondary role, consequent upon interference with normal emptying of the gallbladder. Recently, also, it has been suggested that cholecystitis may develop from some disease of the gastrointestinal tract, such as appendicitis or duodenal ulcer, which, by reflex action, interferes with biliary function. The frequent association of these diseases with cholecystitis is the basis for this theory.

We have become more and more aware of the necessity for establishing definitely the surgical indications in gallbladder disease as experience has shown that most of the poor results follow operations which should not have been done in the first place. It has long been recognized that one irrefutable argument for cholecystectomy is the presence of a stone or stones. Although approximately 10 per cent of patients with gallstones have no appreciable symptoms, this does not mean that the stones are *innocent.* W. J. Mayo, in 1911, more than 40 years ago,

stated that the innocent gallstone did not exist, and his view since has been con-
firmed almost countless times. The possibility of obstruction of the common duct
or acute cholecystitis, or both, is ever present, and once either of these complica-
tions develop the effects can be far-reaching.

From the surgical standpoint, the noncalculous gallbladder calls for a different
approach, one which is negative rather than positive. According to the current
view, in the decision to remove the noncalculous gallbladder, the patient's history
is the deciding feature, supplemented by roentgen evidence of nonfunction. In
contrast to the typical gallstone colic, which is acute and radiates to the right
shoulder region, the noncalculous gallbladder usually is manifested by a more or
less severe and constant aching in the epigastrium incident to edema. Although
at times the pain may become colicky in nature, it is not quite like that associ-
ated with stones. If the pain is persistently severe and is clearly related to the
gallbladder; if the patient is jaundiced or gives a history of jaundice; or if patho-
logic changes in the liver and pancreas are suspected and a total loss of gall-
bladder function is demonstrated, one may feel justified in removing the organ
even though no stones are apparent in the roentgenogram. Not infrequently,
stones will be found in the gallbladder or obstructing the duct, or both. In the
absence of clear-cut symptoms, the demonstration of poor function is no indica-
tion for cholecystectomy. The difficulty may arise from a disturbance of the
sphincter mechanism, which in turn may originate in a psychic disorder, some
abnormal constitutional state, or an organic disease outside the gallbladder.

In either calculous or noncalculous cholecystitis, the symptoms may be at-
tributable in part to an associated duodenal ulcer, intestinal irritability, hepa-
titis, pancreatitis, or perhaps to a hiatus hernia or disease of the heart or kidneys.
Thus, all these contingencies must be investigated. It is especially necessary that
biliary colic be distinguished from right kidney colic and from the pain of coro-
nary occlusion or angina pectoris and acute pancreatitis. A cardiac disorder,
however, is not necessarily a deterrent to removal of the gallbladder; we have
seen patients with a real or supposed heart disease materially improve or recover
completely following cholecystectomy. While chronic pancreatitis may subside
after removal of a diseased gallbladder, the acute type generally is a medical
problem; the two types must therefore be clearly differentiated. Jaundice, if
present, may be intrahepatic or hemolytic in origin rather than due to duct
obstruction, and if so, may be a contraindication to surgery. When the indications
and contraindications are observed and other possibilities are excluded, removal
of the diseased gallbladder often will have a favorable influence upon liver and
pancreatic disease and frequently upon other associated disorders as well.

Probably no single subject in the field of abdominal surgery has been so contro-
versial as the question of the most opportune time for operation in acute cho-
lecystitis. The argument was heard on all sides 25 years ago. It still is heard on
all sides. One cannot help wondering if the difference of opinion regarding the
virtues of early versus late operation, or vice versa, might not be resolved into
one unanimous opinion if it were clearly understood what is meant by *early* and
late. It should mean *early* or *late* in the course of the pathologic process as indi-

cated by the signs and symptoms, the time element being of significance only in its relation to the pathologic changes.

In only a few cases of acute cholecystitis is the process fulminating from the beginning of the attack, requiring immediate operation. As a rule, the inflammatory reaction is at first chemical in nature, being manifested by the sudden or gradual onset of acute epigastric pain with radiation to the subscapular or intrascapular area, mild or moderate leukocytosis and fever, more or less tenderness in the gallbladder region, and abdominal rigidity. The gallbladder may or may not be palpable, and nausea and vomiting may or may not be associated. If operation is undertaken during this stage, the risk usually is slight and recovery rapid. At times, even during these early hours, the pathologic changes may be more advanced than the clinical picture suggests; after 48 hours, however, infection is especially likely to supervene. If the patient is not observed until 48 hours after the onset of the attack and the symptoms are not yet severe, one may assume that the pathologic changes are of mild degree and the attack probably will subside under conservative treatment. If, despite conservative measures, the symptoms and signs increase in severity at any time from the onset or if they persist for as long as 72 hours, surgery is in order without further delay.

In deciding just when to operate in acute cholecystitis, three points are worthy of consideration: First, if the operation is done early in the first attack, the pathologic changes usually will be so slight as to cause little difficulty; second, even though the attack should subside without operation, recurrence may be expected, and every attack carries an increased risk; and third, once infection has developed, it complicates the immediate surgery, should it become necessary, or adds to the technical difficulty of the subsequent operation, even though it may be an interval procedure.

Obstruction of the extrahepatic ducts is the most common complication of cholelithiasis. While the obstruction may be produced by inflammatory edema, stricture, spasm of the sphincter or a tumor of the duct or pancreas, in by far the majority of cases the culprit is a stone or stones.

The pathologic changes produced in the biliary system by obstruction vary widely in both type and extent. The usual effect upon the ducts is dilatation and inflammatory changes, followed by infection and thickening of the walls. Occasionally, the dilatation is saccular and the walls are thinned rather than thickened. When the process extends into the intrahepatic ducts the smaller bile passages and canaliculi become distended and the portal veins and lymph channels are compressed. This situation may lead to atrophy of the liver substance, degeneration of the parenchyma and, finally, to a state of hydrohepatosis. Or, the small ducts may rupture, giving rise to the formation of bile thrombi and necrosis of the surrounding liver tissue. If infection is associated, purulent material may accumulate in the bile passages and, upon rupture of the ducts, may form miliary abscesses. In some cases, the process may go even further; the abscesses may coalesce or enlarge and penetrate into the subdiaphragmatic space or the pleura, or may form a bronchobiliary fistula. In other cases, the ducts may become thickened and fibrotic and the liver substance may also undergo

fibrosis or an extensive cirrhosis, or a combination of these changes. It should be borne in mind that the extent of the destruction in the liver cannot be determined from the patient's symptoms and appearance; even though the liver damage is advanced, the physical evidence may be slight.

The indications for opening the ducts have been extended over the years as we have continued to observe a considerable number of patients who have difficulty from this source after cholecystectomy, and even after choledochotomy. Experience has shown that the more often the ducts are explored, the higher the percentage in which stones are recovered. On the whole, approximately one-fourth of the patients with gallstones will be found to have duct stones, and in the majority the stones will be located in the ampulla where they are most likely to cause obstruction. In addition to those with stones, a small group will have pathologic changes which suggest the need for choledochotomy as a precautionary measure. The criteria which we consider proper for opening the common duct are as follows:

1. Palpable stones in the duct.

2. Abnormal dilation of the duct.

3. A gallbladder containing small stones and a patent cystic duct.

4. Past or present gallstone colic, followed by jaundice, with or without chills and fever.

5. A thick-walled, contracted gallbladder with or without stones.

6. Flocculent bile and sand in the common duct, as determined by aspiration.

In the presence of abnormal dilatation, one often will find the distal end of the common duct occluded by a stone or stricture. In other cases, the duct may have taken over the function of the gallbladder following occlusion of the cystic duct, or, the dilatation may be secondary to a gallbladder infection and thus amenable to relief by cholecystectomy alone. If the gallbladder contains small stones and the cystic duct is patent, it is more than likely that stones will have escaped into the common duct. Colic with jaundice obviously suggests obstruction of the distal end of the common duct by a stone, although other conditions, such as stricture, pancreatitis, a tumor, or spasm of the sphincter of Oddi may produce a similar condition. Also, a thick-walled, contracted gallbladder indicates a long standing disease; even though it does not contain stones, they probably will be found in the duct. Flocculent bile and sand not only call for choledochotomy as a precautionary measure, but these findings frequently are associated with a stone at the outlet of the common duct. When the foregoing criteria are observed, stones should be recovered in at least 75 per cent of the ducts opened.

After evacuation of the hepatic and common ducts by means of a scoop, the patency of the sphincter is determined with a probe or graduated dilators. This is followed by gentle irrigation of the ducts with saline solution. Some surgeons advocate cholangiograms to determine the presence of remaining stones. This procedure is both expensive and time consuming, however, and its value is open to question in the average case. If one is thorough in exploring the ducts, especially the ampulla, and clearly establishes the patency of the sphincter, and then institutes drainage through a T tube, the likelihood of subsequent difficulty

from overlooked stones is remote. The same statements apply to common duct pressure readings to determine the tone of the sphincter.

It is necessary to maintain drainage through the T tube until it can be proved that all the bile passes freely into the duodenum. We have found that this can be proved merely by elevating the tube for a few hours. If one prefers, however, the tube may be clamped. Usually, at least one week is required for spasm of the sphincter to subside completely, permitting normal drainage of the bile.

The most noteworthy developments in surgery during recent years have been the new measures in preoperative and postoperative care. Among these, the antibiotics are preeminent, although we have also gained a new insight into mineral and protein requirements, and the prevention of hemorrhage in jaundiced patients by the use of vitamin K. All of these measures have served to facilitate postoperative recovery and reduce the mortality rate to a remarkably low figure.

Insofar as the actual technic of cholecystectomy is concerned, it seems to me that our chief progress has come to lie in a more acute awareness of the necessity for taking pains. All the antibiotics and other aids to surgery at our command are of no avail in attempts to repair a severed duct, or a stricture caused by the improper use of instruments. Wide exposure to permit complete visualization of the arteries and ducts, and meticulous dissection and careful application of clamps to avoid injury to these structures are vital points in the procedure. It is necessary, also, that the cystic duct be divided neither too far from the common duct nor too near; if too far, then the remnant subsequently may become the source of trouble from retained stones; if too near, the common duct may be kinked and thus partially occluded and ultimately strictured. A safe distance for the ligation is one-fourth inch from the common duct. This should be accomplished without traction on the cystic duct remnant. In addition, if the gallbladder contains stones and a choledochotomy is anticipated, the gallbladder should be removed before the duct is opened; otherwise, stones may be extruded into the duct upon manipulation of the gallbladder.

If identification of the ducts and vessels is difficult, the gallbladder is best removed from above downward, or, a cholecystostomy may be the wiser course. Cholecystostomy also is preferable, as a rule, for elderly, debilitated patients with advanced associated disease.

COMMENT

The status of gallbladder surgery, as of any other type of surgery, is determined by the individual surgeon's recognition and observance of the various factors which enter into the success of the operation. Reduced to the simplest terms, the factors which largely influence the success of gallbladder surgery are: (1) The duration of the disease and the extent of damage to other structures; (2) definite diagnosis and definite indications for operation; and (3) painstaking, atraumatic operative technic, with ample exposure of the ducts and vessels and removal of all stones.

While other considerations, including the preoperative and postoperative care and choice of the procedure, have a material bearing upon the outcome, the prob-

lems inherent in these three provide the real test of the surgeon's acumen and ability. If, however, unless plainly contraindicated, cholecystectomy were done routinely as soon as the presence of stones was discovered, many of these problems would cease to exist. Perhaps the most progressive feature of gallbladder surgery during the past 25 years has been an increasing appreciation of the advantages of early removal of the gallbladder containing stones.

TREATMENT OF CHRONIC RELAPSING PANCREATITIS

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Chronic pancreatitis first was described in the literature by Fitz in 1889. Since that time the syndrome of chronic relapsing pancreatitis has been neglected and almost forgotten until recently. During the past 10 years it has assumed a rather important place as a diagnostic and therapeutic problem. The condition has been mistaken for many other disease entities. Frequently patients with chronic pancreatitis are branded as neurotics, hypochondriacs, biliary dyskinesias, pylorospasms, gastro-enteritides or intestinal allergies.

The entire subject of chronic relapsing pancreatitis is too broad to be considered in its entirety here, but we will outline briefly the etiologic factors involved, the signs, symptoms, diagnosis, both laboratory and roentgenologic, the operative findings and the treatment.

ETIOLOGY

Seven principal etiologic factors are involved:

1. Biliary obstruction due to stone, tumor, spasm, duodenal diverticulum, duodenal mucosal edema, and papillary stenosis.

2. Regurgitation of bile due to the formation of a common channel by the pancreatic and common bile ducts, reported as occurring in 50–60 per cent of cases.[4, 7]

3. Trauma, accidental or surgical.

4. Infections of the biliary tree with secondary extension into the pancreas. This sometimes develops after scarlet fever, mumps, typhoid, or an acute septic process.

5. Alcohol, drugs and anesthetics.

6. Circulatory factors such as stasis, hypertensive apoplexy, thrombosis, and embolism.

7. Primary infections of the pancreas.

8. Psychogenic stress with hyperactive vagus, and therefore the production of increased pancreatic secretion and spastic occlusion of the pancreatic ducts.

The four etiologic B's that apply to acute pancreatitis also are involved in chronic relapsing pancreatitis. Therefore, it might be appropriate to mention them here to aid the memory. They are bile, blood, bacteria and booze.

The disease may occur at any age. Cases have been reported occurring in patients between the ages of 10 and 75 years, the average[13] age being about 50 years. Gallbladder disease occurs in approximately 50 per cent of the patients. This brings up some very interesting discussion. Some believe that pancreatitis

frequently precedes the biliary disease and is not always the result of it. This we believe often occurs, since in our experience we have found no biliary disease in 50 per cent of our patients. Alcohol is a very important etiologic factor, in acute pancreatitis and in chronic relapsing pancreatitis, and should be given special emphasis.

SIGNS AND SYMPTOMS

The only consistent symptom of chronic relapsing pancreatitis is epigastric pain. The pain usually is boring in character, girdle-like passing around the trunk, and radiating to the back. It is severe and lasts for hours, days or weeks, after which the patient may have a long or short symptom-free period. The pain is constant and not colicky as is usually seen in gallbladder disease. Vomiting may or may not occur, but nausea frequently is present. Occasionally, patients will have mild jaundice. Usually there is intolerance for fatty foods, and indigestion with marked weight loss. A patient may reach the stage where he is afraid to eat, which produces weight loss often exceeding 30 pounds. In advanced cases patients may have *pancreatic stools* containing a large quantity of undigested fat and muscle fibers, or there may be frank steatorrhea and mild diabetes, the diagnosis then being relatively simple.

PATHOLOGY

The microscopic morphology of chronic pancreatitis is variable. The glandular tissue may be preserved or reduced due to preceding destruction. In some patients the hormonal islet tissue may be spared but often it also is damaged. Metaplastic and cystic changes of the ducts are common.

Chronic inflammatory infiltration, fibrosis, necrotic remnants in glandular and surrounding fatty tissue and calcification can be present together. However, more commonly two types can be recognized: the fibrotic-calcific pancreas and the pancreas with chronic interstitial inflammation mostly with cellular elements. The former follows usually less severe cases of pancreatic necrosis while the pathogenesis of the latter is not quite clear. In either case the pathologic lesion of chronic pancreatitis is not always diffuse. It very often consists of spotted areas which may alternate with normal pancreatic tissue.

DIAGNOSIS

The most important factor in making a diagnosis of chronic relapsing pancreatitis is a high index of suspicion, always considering this condition in the differential diagnosis of any upper abdominal pain. The diagnosis usually is made by a combination of physical examination, laboratory, roentgenologic findings, and above all a pertinent history. The patients usually have had studies to rule out gallbladder disease, peptic ulcer, kidney colic and other common disorders. Fifty per cent of the patients have had previous surgery. In these patients the gallbladder and the appendix are the organs most frequently attacked with no relief of symptoms. The pain frequently requires narcotics and the patient soon is branded an addict, when in reality the pain is very real.

Patients labeled as neurotics, postcholecystic syndrome, biliary dyskinesias, or just plain hypochondriacs should be carefully studied to rule out chronic pancreatitis and be given the benefit of surgical treatment when indicated.

On physical examination deep tenderness is present in the epigastrium. There usually is evidence of marked weight loss. The serum amylase or lipase may be elevated during an exacerbation but this occurs in only about one-third of the cases. The roentgenologic picture may show calcification of the pancreas, this being true in 2 of our 13 patients. The finding of pancreatic stones is quite conclusive.

At times one must rely only on the above clinical findings to make a diagnosis, an exploratory laparotomy being necessary to be conclusive. At operation the pancreas generally is found to be indurated and nodular and sometimes cystic, either enlarged or smaller than usual, depending upon the degree of inflammation and the stage of the disease.[2] Some of our patients were given splanchnic blocks with complete relief of symptoms prior to operation, a procedure which may help to determine the advisability of corrective surgery. In addition to ruling out such common conditions as biliary disease, gastric or duodenal ulcers, carcinoma, and pylorospasm, such entities as sprue, syphilitic crisis and true psychoneurosis must be considered.

TREATMENT

Medical treatment should be tried first and should consist of proper diet, the interdiction of alcohol, use of antispasmotics, antisecretory drugs, enzymes and vitamins. If these fail surgery should be considered.

Many operations have been devised for the treatment of chronic pancreatitis.[3, 4, 5, 6, 7, 8, 9, 10, 11, 12] Some of the procedures that have been used with varying degrees of success are as follows:

1. The correction of biliary disease.
2. Relief of pressure on the duodenum and sphincter of Oddi by cholecystostomy.
3. Drainage of pancreatic cysts or abscesses.
4. Removal of calculi from the pancreatic ducts.
5. Anastomosis of the gallbladder to the bowel or stomach.
6. Drainage of the common bile duct.
7. Pancreatectomy—partial or total—or pancreatostomy with removal of stones.
8. Choledochojejunostomy (Roux Y).
9. Sphincterotomy, which has been a failure in the absence of biliary disease.
10. Sympathectomy and splanchnicectomy.
11. Vagotomy.
12. Gastric resection with or without vagotomy.
13. Celiac ganglionectomy and bilateral splanchnicectomy.

Of the procedures mentioned above the most popular are sphincterotomy[4] and the Roux Y procedure with choledochojejunostomy.[2]

There has been very little in the literature about the use of celiac ganglionectomy and bilateral splanchnicectomy[6] in the management of chronic pancreati-

tis. This we propose as a method of treatment. The splanchnicectomy relieves the pain and the spasm of blood vessels and the ducts. The added celiac ganglionectomy enhances this relief and helps prevent the postganglionic nerve regeneration. The added physiologic response is increased blood supply to the pancreas. This method seems to be logical and is not as *mutilating* as other more radical operations, such as pancreatectomy, which leaves the patient a pancreatic cripple.

PROCEDURES

The Conventional Procedure. The abdomen is first thoroughly explored for any other disease. If gallbladder disease is present, cholecystectomy is done and if the common bile duct is dilated a sphincterotomy and T tube drainage[7] is also done after carefully searching for gallstones and common bile duct stones. When the common bile duct is dilated sphincterotomy is done regardless of the presence or absence of stones. In the usual approach the gastrohepatic ligament is entered just to the left of the portal vein. The crux of the diaphragm and the aorta at the level of the celiac axis are identified, exposing the celiac ganglia, which surround the axis. The greater and lesser splanchnic nerves are isolated as they pass through the crux of the diaphragm to enter the ganglia. Both ganglia are removed by blunt and sharp dissection and the splanchnic nerves are cut 2 to 5 cm. proximal to their entrance into this sympathetic plexus (fig. 1).
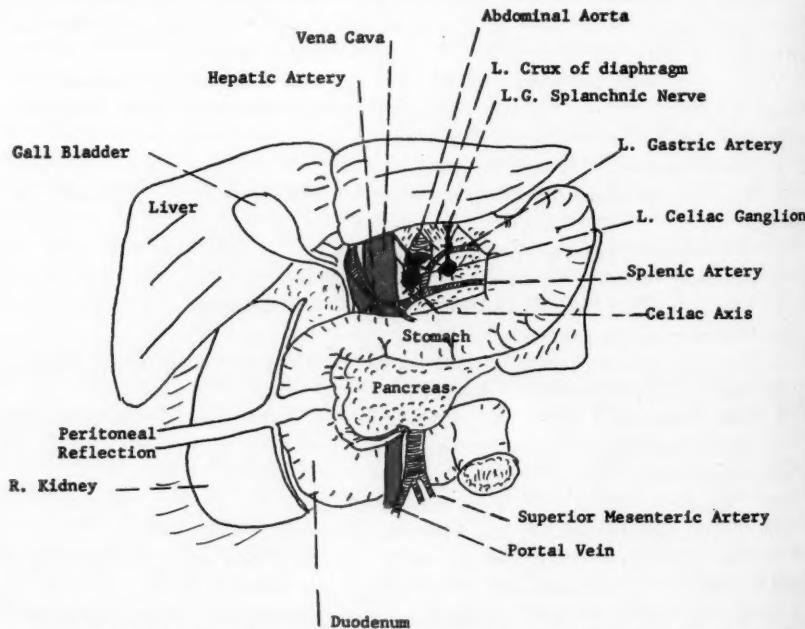
FIG. 1. Diagram demonstrating the normal position of the celiac ganglia and splanchnic nerves, and the routine supragastric approach.

A different surgical approach to the celiac ganglion has been devised by one of us (F. L. C.). In obese patients, those with a short gastrohepatic ligament, high lying pancreas, or short celiac axis, the conventional approach may be very difficult. In these patients, the lateral peritoneal reflection of the second portion of the duodenum is incised and the duodenum and adjacent pancreas are rotated anteriorly and to the left, exposing the underlying vena cava and the right renal vein and artery. The portal vein and common duct are rotated with the duodenum. As the structures are rotated medially, the aorta, the right suprarenal gland and vessels, and the right celiac ganglion are exposed.

The ganglia lie around the celiac axis, at the level of the upper margin of the first lumbar vertebra. The one on the right usually lies behind the vena cava. From the ganglia, sympathetic fibers radiate to the upper abdominal viscera. Above, the greater, lesser and least splanchnic nerves usually can be isolated and transected. Care must be taken to prevent severance of the lumbar veins which will bleed freely. The left splanchnics are more difficult to expose and lie lateral and above the left ganglionic mass (fig. 2).

It is of prime importance to remove all ganglionic tissue with a portion of the splanchnics to insure complete relief of pain, because the celiac ganglia also receive branches from 1, 2, and 3, lumbar nerves. Often the superior mesenteric ganglia also are removed to insure relief. This approach, although it may appear to be complicated and dangerous, may be made rapidly and without great surgi-
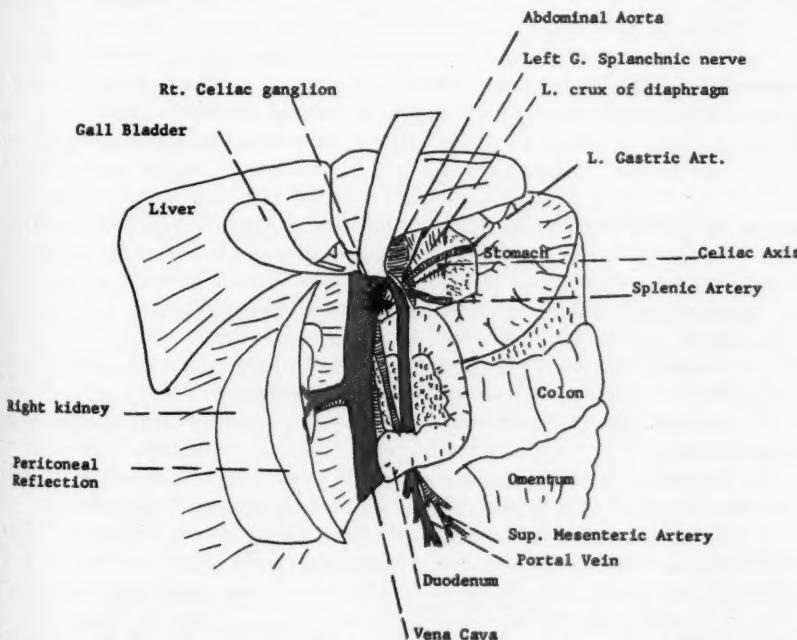
Fig. 2. Diagram demonstrating the reflected duodenum and pancreas with exposure of the celiac axis and ganglia.

TABLE I

Tabulation of patients treated by ganglionectomy and splanchnicectomy

Patient	Age	Sex	Biliary Disease at Time of Our Surgery	Previous Biliary Surgery	Biopsy For Pancreatitis	Pancreatic Calcification	Results
N.C.	53	F	Yes	Yes	Positive	No	Good
C.E.S.	58	M	No	No	Positive	Yes	Good
L.W.	45	F	No	Yes	Positive	No	Good
G.B.	56	F	No	Yes	Positive	No	Good
C.S.	37	M	No	No	Negative	No	Good
L.A.	57	F	No	Yes	Not taken	No	Good
A.W.	36	F	No	No	Not taken	Yes	Good
P.O.	32	F	No	Yes	Not taken	No	Good
M.G.	29	F	Yes	Yes	Not taken	No	Poor
F.C.	62	F	No	Yes	Not taken	No	Fair
L.W.	24	F	No	No	Positive	No	Undetermined*
M.D.	59	M	No	No	Positive	No	Undetermined*

* Patients done recently, not yet evaluated.

cal difficulty. In the hands of the authors this approach in many patients is easier and more rapid, and less bleeding is encountered than when the gastro-hepatic approach is used.

RESULTS

The first of our patients was operated upon two years ago and the last patient approximately two months ago. Two of the patients had been in various hospitals over a period of three years and were branded neurotics. They now have been completely symptom free since surgery. Nine of our patients had complete relief of symptoms, 1 had partial relief, and 1 was a failure. Two of these patients showed calcification of the pancreas and 6 others had biopsies which were reported as chronic pancreatitis (table I). All obvious cases of chronic pancreatitis were not biopsied because of the added dangers of hemorrhage and fistula formation.

The advantages of the procedures are summarized as follows:[7]

1. The biliary tract is inspected, being easily accessible.

2. Any stones missed at the time of previous surgery can be removed.

3. A biopsy of the pancreas may be done if indicated.

4. The pancreas can be directly inspected and palpated for cysts, tumors or inflammation.

5. A combined splanchnicectomy and celiac ganglionectomy can be done with ease and combined with sphincterotomy and biliary surgery if indicated.

6. Since the smooth muscle of the intestines, blood vessels, biliary and pancreatic ducts are innervated by the sympathetic nerves, splanchnicectomy and celiac ganglionectomy abolish spasm of the muscles and blood vessels particularly in the sphincter of Oddi.

7. Celiac ganglionectomy and splanchnicectomy increase the blood supply to

the pancreas, aiding the healing of the inflamed pancreas and tend to terminate the disease.

8. Pain, whether caused from pancreatitis or not, empirically is relieved and the patient exists comfortably.

The most important single factor resulting from this procedure has been the relief of pain. Inflammation causes pain, spasm and constriction of blood vessels. Splanchnicectomy and celiac ganglionectomy stop pain, alleviate spasm, increase blood supply and, therefore, promote healing.

SUMMARY

Thirteen cases of patients who had chronic relapsing pancreatitis, treated by celiac ganglionectomy and bilateral splanchnicectomy, with or without sphincterotomy and biliary surgery, have been presented.

The diagnosis and etiologic factors are discussed.

Two surgical approaches to the celiac ganglia and splanchnic nerves have been described.

The rationale of the procedure is: A. All pain fibers to the pancreatic area, duodenum and biliary tract have been interrupted by the splanchnicectomy; B. by celiac ganglionectomy and splanchnicectomy, spasm of the sphincter of Oddi and of the vessels supplying the pancreas have been relieved; and C. by the splanchnicectomy the blood supply to the pancreas is increased, aiding healing of that organ.

A different surgical approach to the celiac ganglia and splanchnic nerves is proposed for use in selected patients.

The results of the cases of thirteen patients who were treated by celiac ganglionectomy and bilateral splanchnicectomy with or without sphincterotomy and biliary surgery are presented.

CONCLUSIONS

Chronic pancreatitis must be considered in the differential diagnosis of upper abdominal pain.

Chronic pancreatitis is not necessarily associated with biliary disease.

Celiac ganglionectomy and bilateral splanchnicectomy, with the correction of biliary disease, if it is present, is an effective method of treating chronic relapsing pancreatitis. The follow-up period on these cases is too short, we realize, to be didactic but at the present time we believe that this is a rational and effective approach to the problem and warrants further investigation.

The retroduodenal-retropancreatic approach to the celiac ganglia appears to be of aid in those patients who are inaccessible by the orthodox approach.

The transperitoneal approach to the celiac ganglia and splanchnic nerves permits exploration of the abdominal cavity.

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THE DIAGNOSIS AND MANAGEMENT OF THYROID DISEASE

A PATHOLOGIC EVALUATION

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During the past 25 years there have been many changes in the diagnosis, treatment and general evaluation of thyroid disease. Some of the changes have been due to development of new forms of treatment. Many of the changes, however, have been due to a better basic understanding of thyroid physiology and thyroid pathology. I should like to consider here some of the current concepts of thyroid pathology and how such concepts aid in clinical treatment.

Although there have been some interesting developments in the diagnosis and treatment of Graves' disease, adenomatous goiter and thyroiditis, such as the use of thiouracil and radioiodine, it is the evaluation of tumors of the thyroid that is of particular interest to the surgeon and pathologist. The discussion here will deal with certain questions regarding thyroid tumors which are most frequently asked of pathologists by surgeons.

1. *What is a simple working classification of thyroid tumors?* One important step in the modern evaluation of thyroid disease has been the development of a more uniform and a simpler pathologic classification. The classification which we currently use is seen in table I.[6] It is divided into four major groups: primary hyperplasia or Graves' disease, adenomatous or multinodular goiter, thyroiditis, and tumors.

Tumors of the thyroid present peculiarities of growth and structure which make their classification more varied and difficult than tumors arising in most tissues of the body.[7] Since any classification by the surgical pathologist should be a helpful one which will assist in the better clinical treatment of an individual case, the current classification we use is simple and meant to be of clinical use.

Benign tumors of the thyroid, for practical purposes, are all adenomas. While in the past adenomas have been extensively subclassified, the current classification divides them only into follicular and papillary types, the latter being extremely uncommon. This simple descriptive division seems adequate and does away with the confusion from terms such as *fetal*, *embryonal*, and *trabecular*.

The distinction between adenomatous goiter and true adenomas still causes some confusion both to the clinician and to the pathologist. True adenomas are almost always solitary and are real neoplasms, as the name *adenoma* implies, whereas the nodules of an adenomatous goiter are multiple and are manifestations of hypertrophy, hyperplasia, and fibrosis. The distinction between the two is of importance, since it is currently believed that only the true neoplasm has an appreciable precancerous significance.

TABLE I

Primary hyperplasia
Adenomatous Goiter
Thyroiditis
Tumors
   Benign (adenomas)
     Follicular
     Papillary
   Malignant (carcinomas)
     Undifferentiated
     Differentiated
      Follicular
      Papillary

Practically all malignant tumors of the thyroid gland are epithelial and are thus carcinomas. About 25 per cent of them are undifferentiated. These are highly malignant, usually inoperable, and, unfortunately, usually incurable.

The differentiated carcinomas actually are the malignant counterparts of adenomas and subsequently are classified into follicular or papillary types. The papillary carcinoma is much the more common of the two types (conversely with adenomas!).

2. *What is the pathologic meaning of the term "invasive adenoma"?* In addition to the general classification of thyroid tumors, an attempt is made in the differentiated tumors to establish the extent of the cancer pathologically and thus to assist in the evaluation of further treatment necessary in individual cases.[4] Many carcinomas of the thyroid appear grossly as adenomas and it is only after microscopic examination that they are found to be malignant by some rather minimal, but nevertheless definite, criteria of malignant change. For example, a tumor appearing to be a gross adenoma, might, on microscopic examination, show invasion of lymphatic vessels, of blood vessels, or of its capsule, and therefore properly would be called a carcinoma on the basis of microscopic examination. Obviously, however, such cancers are in a different stage of their development from tumors which can be diagnosed as cancer not only microscopically, but from the gross appearance as well. The group of adenomas with invasion, therefore, represent an early or minimal stage of carcinoma and one that should be more readily curable with less radical treatment. The significance of blood vessel invasion in assisting in making the diagnosis of malignant tumors was emphasized by Graham in 1924[2] and has been of increasing use to pathologists in helping to determine whether or not any given lesion is benign or malignant.[3] It should be emphasized that in almost all cases, when blood vessel invasion is found there are other evidences of malignant change as well.

3. *What is the present status of lateral aberrant thyroid tumor?* Twenty-five years ago it commonly was believed that the occurrence of aberrant thyroid tissue in the lateral neck was frequent and that tumors arising from such aberrant tissue likewise were frequent. Following numerous case studies done by different individuals during the last 25 years, it has become apparent that this concept is

incorrect.[8] Lateral aberrant thyroid tissue does occur occasionally, but tumors arising from such tissue are indeed very rare. In practically all instances, the tumor suspected to be of lateral aberrant thyroid origin represents in reality a metastasis to a lymph node from a small focus of carcinoma in the main gland itself. This focus may be extremely small and may not be visible or palpable. Nevertheless, the finding of thyroid type tumor in the lateral neck necessitates a removal of the thyroid lobe on the same side.

4. *What is the current status of radioiodine in the treatment of thyroid tumors?* The best rule of thumb regarding whether or not radioiodine will be usable in a given tumor, is whether or not the tumor contains colloid. If it does, it has a good chance of taking up radioactive iodine sufficiently to be used in treatment. Only about 50 per cent of thyroid tumors will take up sufficient radioiodine so that it can be used for treatment.[1] This means that, for the most part, these are the well-differentiated follicular carcinomas containing considerable colloid. It means, therefore, that these are lower grade tumors and thus tumors that usually can be treated well by surgical procedures. The unfortunate aspect of radioiodine treatment of thyroid tumors is that it does not help where help is the most needed; that is, in the highly undifferentiated tumors that are not amenable to treatment by surgery.

5. *What nodules of the thyroid are of surgical importance and warrant excision?* This is the most important and most frequent question asked of a pathologist regarding thyroid tumors. In our experience, the nodules of a multinodular goiter are not precancerous, or if so, only to a very minimal degree. It is the solitary nodule that is of especial interest and importance. A solitary nodule has the best chance of being a true neoplasm—an adenoma. The possible potentialities of an adenoma are several: (1) as any benign tumor, it may slowly increase in size until it causes either disfiguration or pressure on some important structure in the neck; (2) the nodule may undergo various types of retrogressive changes such as hemorrhage and infarction, which cause it to increase rapidly in size, and thus simulate a rapidly growing cancer; (3) the adenoma may become hyperplastic to such a degree that the patient has clinical hyperthyroidism. This is extremely rare; only about 1 per cent of true adenomas are clinically *toxic*. The *toxic adenoma* usually is a nodule which is not an adenoma at all but an adenomatous goiter with secondary hyperthyroidism; (4) the adenoma may become cancer. This possibility has been much discussed and has been suggested as the main reason for the removal of a solitary nodule, the theory being that it may become malignant if allowed to remain behind. Proof of such a transition of adenoma to carcinoma is not easy for the pathologist to demonstrate, although it often seems obvious to the clinician. While pathologists accept the proposition that such a transition may occur, there is an increasing suspicion that many carcinomas of the thyroid begin as carcinomas from their inception without necessarily going through a benign tumor phase on their way to becoming cancer. Particularly, this seems to be the case in the papillary carcinomas, since benign counterparts of papillary carcinomas are extremely rare.[5]

As far as the danger of cancer in a solitary thyroid nodule is concerned, the

most useful clinical evaluation from the pathologist's point of view is to accept such a nodule as a diagnostic problem. In other words, any suspicious nodule of the thyroid should be removed primarily to see if it is already cancer and only secondarily to prevent its possible transition to cancer at some future date. To the pathologist, the evaluation of thyroid nodules is quite similar to the evaluation of nodules in the breast. The nodule which should be excised is the one which is solitary or the one which is unusual because of rapid growth or other changes. Most important, the nodule, when removed, should be removed primarily for pathologic examination rather than having as the main goal the prevention of some future transition to cancer.

CONCLUSIONS

There are many other important problems still to be solved in thyroid disease. The diagnosis, treatment, and, therefore, the pathologic study of thyroid disease, is undergoing continual change. Since the thyroid gland is an endocrine organ, the concepts regarding its diseases are changing faster than those regarding many other organs of the body. Ever increasing knowledge of thyroid physiology and pathology will, therefore, in the 25 years to come, undoubtedly produce as many new concepts and as many changes in evaluation as have been seen in the last 25 years.

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DIFFERENTIAL DIAGNOSIS AND MANAGEMENT OF THE SENILE PROSTATE SHOWING TWENTY-FIVE YEARS OF PROGRESS

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It is indeed a great honor and also a great responsibility to appear on this Silver Jubilee Assembly of the Southeastern Surgical Congress. It also is a most difficult assignment to take the place of Dr. Hugh H. Young who addressed this assembly 25 years ago. I have read his paper with great interest and if he were here at this meeting it would be to him, I am sure, a source of deepest satisfaction to realize the great progress that has occurred in the specialty of urology during these 25 years. These 25 years have witnessed the perfection of excretion urography, the introduction of antibiotics, the perfection of the operation of transurethral resection and the development of endocrine methods in the management of carcinoma of the prostate. All of these steps have been of the utmost significance in the progress of the specialty of urology; have played a most important part in the differential diagnosis and in the management of diseases of the prostate gland and have resulted in outstanding reduction of both morbidity and mortality rates and in the relief of human suffering.

Progress in the differential diagnosis of diseases of the prostate has not been as outstanding as in the other phases which I have mentioned. Diagnostic methods were well advanced 25 years ago due to the perfection of various types of cystoscopes and increasing familiarity and skill in their use throughout the urologic profession. The importance of careful examination of the prostate by digital palpation was, of course, thoroughly realized 25 years ago, as it is today, and by this method the various degrees of hypertrophy and especially the early recognition of malignant disease was thoroughly emphasized then, and the importance of rectal examination in all men over the age of 40 years was self-evident, but at that time the necessity for this examination was not given the importance and emphasis that is placed upon it today. The use of the various cystoscopes, which indeed were highly perfected 25 years ago through the cooperation and ingenuity of instrument houses throughout this country, has been further improved to allow a very complete diagnostic survey of the prostate gland with the study of the effects of any obstruction it may have been causing, so that then, just as today, we were able to make an accurate analysis of the pathologic changes in the prostate; to make a fairly accurate estimate of its size and the degree of obstruction which it was causing and therefore to choose, if operative relief were deemed necessary, the proper method by which the obstruction should be corrected.

Excretion urography was introduced in 1929 by Zwick, and through the following years programs of urologic meetings were filled with studies on the value

of this method in the differential diagnosis of disease of the upper urinary tract. This procedure now is in general use in the study of prostatic disease to estimate the degree of any back pressure on the upper urinary tract or other disease which might complicate the treatment of the patient. Indeed, a radiologic study by intravenous injection of radiopaque chemicals often will give a complete study of the urinary tract without the necessity of cystoscopic examination, particularly if delayed roentgenograms are obtained one or two hours after injection of the material, and particularly if the patient is asked to void before the latter films are taken. Urography will give accurate information as to the amount of residual urine present, which always has been and always will be a most important consideration in leading to the decision that surgical intervention is necessary.

During recent years there has arisen a great interest in the possibility of complete excision of malignant disease of the prostate by the radical operation. Dr. Young did the first radical perineal prostatectomy in 1904, having been stimulated at this time by the work on radical mastectomy of Dr. W. S. Halsted, Professor of Surgery at the Johns Hopkins Medical School at that time. It is of historic interest to note that Dr. Halsted was Dr. Young's first assistant at this first radical operation for early carcinoma of the prostate. For many subsequent years the possibility that early disease of the prostate could be completely removed by radical operation was largely ignored. Only a few urologists, who had not been pupils of Dr. Young, did the operation. During the past few years, however, there has been a marked revival of interest in the radical operation in part, at least, due to the popularity of retropubic prostatectomy for simple hypertrophy, and the fact that this approach can be utilized for radical prostatectomy by those who are not sufficiently familiar with the perineal technic.

Coincident with this revival of interest in early diagnosis newer methods for this purpose have been suggested. The value of perineal exposure of the prostate and excision of the area of suspicious induration under direct vision and its immediate study by frozen section were given early emphasis by Dr. Young, and this method has indeed stood the test of time.

Punch biopsy, as a method of diagnosis of a suspicious area in the prostate found on rectal examination, has been proposed, and numerous surgeons have presented instruments by which tissue can be obtained. Certain serious objections can be made to this method. In the first place, a needle is introduced through the perineum into the suspected area, and this area may be missed easily in this procedure. Also, in most instruments the amount of tissue removed is small and great difficulties of interpretation of the specimen removed may arise, but, above all, this procedure definitely is unsurgical. Let us visualize it. A sharp instrument is introduced into an area of actively growing carcinoma and is then withdrawn through the tissues of the perineum. It would seem certain that malignant cells on the needle would be left behind in the tissues through which the needle is withdrawn. The fact that malignant cells can be transplanted is only too well known. Deming has succeeded in producing active growth of cells from carcinoma of the prostate in an entirely foreign soil—the anterior chamber of the rabbit's eye.

Unfortunately many urologists have had the experience of recurrence of malignant disease in the recti muscles after suprapubic operations upon cancer of the bladder, and the only answer to this is implantation of viable malignant cells. Leadbetter recently has reported a case of implantation of prostatic adenocarcinoma in the needle tract through which a punch biopsy previously had been obtained several months before. Dutra and Gerazi also recently published the report of a patient in whom implantation of carcinoma of the lung occurred in the tract through the chest wall through which a needle for a biopsy had been introduced. We have encountered no such case at the Brady Urological Institute because the punch biopsy has been considered an unsurgical procedure and this technic has never been used.

In the last few years great interest has been aroused by the work of Papanicolaou on cytologic diagnosis of cancer, and numerous studies have been reported in which this method has been proposed for early diagnosis of carcinoma of the prostate. To obtain specimens for cytologic examination it is, of course, necessary to express forcibly the contents of the prostate into the urethra and the resultant specimen then is studied according to Papanicolaou's technic. For the interpretation of specimens the services of a trained cytologist are necessary, and in published reports on this subject a fairly high percentage of both false-positive and false-negative examinations can be found. Aside from this objection, it is difficult to understand how the method of obtaining the specimen can be justified. It is well known that extension of prostatic carcinoma occurs through the perineural lymphatics. To obtain sufficient material for cytologic examination, forceful manipulation of the gland must be made and it seems most probable that in this procedure malignant cells might be forced easily into the perineural lymphatics. L. C. Knox in 1922 and M. C. Marsh in 1927 demonstrated experimentally that forceful massage to experimental tumors in mice produced a 22 per cent increase of metastasis as compared with the control series of animals. Therefore, it might be said that no method for the early diagnosis of carcinoma of the prostate has been advanced in the last 25 years which can approach the reliability of perineal exposure of the gland and frozen section biopsy.

Ormond Culp recently has summarized this question as follows: "It has been axiomatic that precise diagnosis is a prerequisite of rational therapeusis. Many ingenious technics have been devised for histologic study of palpable nodules in the prostate, but only perineal exposure of the gland affords direct access to the entire posterior lamella. Biopsy by way of this route, therefore, should have the greatest potentiality in indicating unequivocally the cases suitable for complete eradication of malignant disease by radical perineal prostatectomy. Frozen section examination and simultaneous prostatectomy of appropriate type has no parallel in other diagnostic or therapeutic schemes."

The importance of routine rectal examination, which is the only method by which early suspicious nodules can be detected, has become recognized generally throughout the profession. There have been reports in the literature stating that only 5 per cent of cases of clinical carcinoma of the prostate are suitable for the radical operation. In a 5 year study of all patients seen at the Brady Urological

Institute in whom a diagnosis of carcinoma of the prostate was made, 22.7 per cent were found suitable for the operation which was done. The relatively high percentage of patients suitable for radical operation in this series of cases can be explained by the fact that many of these patients were sent expressly for the purpose of radical operation by the physician who had detected a nodule of suspicious induration on rectal examination.

The studies at the Walter Reed Hospital are even more illuminating. It is well known, of course, that in all military personnel over the age of 40 years rectal examinations at annual physical examinations are mandatory. Colonel Kimbrough has reported that of all patients with a diagnosis of prostatic cancer seen at the Walter Reed over a 5 year period, 54.7 per cent were found suitable for the radical operation and this procedure was done. These figures are of sufficient importance to emphasize the great value of routine digital examinations if cases of prostatic carcinoma are to be diagnosed at a stage early enough for complete surgical eradication.

Before the radical operation is done certain criteria must be rigidly followed, and it is believed that adherence to these principles is largely responsible for the absence of mortality in our present series and for the improvement in functional end results. These criteria are as follows: First, the suspected induration must not extend beyond the capsule of the prostate into the membranous urethra or extensively involve the fascia around the seminal vesicles, and the whole gland must be freely movable. Second, there must be no demonstrable metastasis, either on physical examination or more particularly by roentgenologic studies, and the acid phosphatase determination should be within normal limits. Third, the patient should be a good surgical risk and, most important, his life expectancy must be good. Prostatic cancer is a slowly progressive disease in most patients and we cannot condemn too strongly the radical operation in an elderly man whose life expectancy obviously is limited. Also, in older individuals muscle tone is impaired to some degree at least and the functional result following the operation will never be as satisfactory as when the procedure is done on younger men with good muscle tone. It has, therefore, been our custom to reserve the radical operation for patients under 70 years of age, but this rule is by no means didactic as exceptions always will be found on either side of this limit.

Preoperative care of patients afflicted with prostatism has undergone little change in the past 25 years. The phenolsulphonphthalein determination has survived the test of time, and by this simple method and the studies of the blood chemistry, optimum time for surgical intervention, by whatever method may be chosen, can be fairly accurately determined. The value of preoperative catheter drainage in obstructive cases also has stood the test of time, but the introduction of the Foley catheter has made the management of this phase of preoperative care much simpler and much more comfortable for the patient. The routine employment of the sulfonamides or antibiotics during the care of such patients in whom catheter drainage is deemed necessary has been a striking advance in the prevention of infections which during this stage were so common. Before the introduction of antibiotics the occurrence of severe urethritis and periurethral

abscess as well as epididymitis were only too prevalent, but when these agents are used judiciously infection is extremely rare so that mortality and morbidity rates have been greatly reduced.

It is not the purpose of this presentation to enter into a discussion of the technical details of the various operations by which obstructive disease and carcinoma of the prostate may be treated. For the relief of benign obstruction, in addition to perineal prostatectomy and suprapubic prostatectomy, the retropubic approach has been introduced within the last 10 years and is being used by numerous surgeons who have obtained excellent results with this operation. Transurethral resection 25 years ago was in a more or less trial stage, but at present the great value of this procedure is now well recognized and it occupies a foremost place in our therapeutic armamentarium. It is interesting that this procedure has been a development of the punch operation which Dr. Young introduced in 1911. When this procedure was first employed it was used as a cold cutting instrument without any cystoscopic attachment and was of particular value in the relief of such conditions as contracture of the vesical orifice and median bar formation. The cold cutting principle still is employed by the Thompson resectoscope with, of course, the addition of cystoscopic vision and methods for the electrocoagulation of bleeding points. Truly, it might be said that Dr. Young's early cold punch was the ancestor of the many instruments which have been perfected in recent years for transurethral resection of prostatic tissue.

While it is true that all urologists should be familiar with all methods at present available for the treatment of prostatic disease, either benign or malignant, it is inevitable that in many individuals their training should be directed toward the perfection of one particular method. There is no question that in trained hands all of these methods give excellent results, but no one method can be applied to all types of prostatic disease. It is becoming more generally recognized throughout the country that the transurethral procedure should be limited to operations on glands in which it might be estimated that 50 grams of tissue must be removed to give a good result. This statement probably will be criticized by some surgeons who have been trained especially, and have become particularly skillful, in this technic. Familiarity with transurethral resection is an absolute necessity in the urological world today as results can be simply obtained by this procedure in patients in whom attempted enucleation might result in unnecessary trauma to the area operated upon. This statement applies particularly to fibrous glands involved by chronic inflammatory disease in which a good line of cleavage often can not be obtained. Transurethral resection also has been a most valuable procedure for the relief of obstruction due to malignant disease too far advanced for the radical operation. Previous to 25 years ago many of these patients were condemned to a permanent suprapubic cystostomy with persisting hemorrhages and incrustation of the tube. Transurethral resection often provides for these unfortunate individuals a relatively normal restoration of urinary function until their death from ascending infection or generalized carcinomatosis.

Following all surgical procedures, whether for benign or malignant disease, drainage of the urinary tract either by catheter or suprapubic tube is essential,

and the system of any drainage in use must be kept under meticulous observation to insure that drainage is free and not obstructed. If drainage depends upon a single tube, necessity for continual observation is doubled because such drainage can be easily obstructed by clots with resultant danger from overdistension of the bladder, discomfort to the patient and the possibility that bleeding may be increased. The two-way system of drainage which is in use in our clinic, both in perineal and suprapubic prostatectomy, offers an especial source of safety during the immediate postoperative period in that if one tube should be obstructed, drainage will be continuous through the other tube without any bladder overdistension or the risk of extravasation.

It is interesting to read in Dr. Young's presentation 25 years ago: "On the return of the patient to the ward he is given a submammary infusion of salt solution and vigorous internal hydrotherapy is carried out through his convalescence. The object of this measure is not only to keep the kidneys active but also to combat infection by washing purulent deposits from the bladder. If the patient should develop fever I often employ intravenous therapy, generally 15 cc. of a 1 per cent solution of mercurochrome."

It is interesting that at this time Dr. Young was searching for an efficient method of combatting postoperative infections and his advocacy of the intravenous use of 1 per cent solution of mercurochrome was a result of this objective. Postoperative infection of the blood stream always had claimed a definite mortality rate, even though it was low. With our present methods of preoperative and postoperative use of antibiotics the occurrence of severe infection of the blood stream is rare indeed and the healing of wounds has been greatly expedited. In fact, it might be said that the severe incrusted wound infection, formerly seen in suprapubic incisions and also in perineal and occasionally in kidney incisions, is a thing of the past.

It was only a few years after Dr. Young's time that the work of numerous research laboratories on shock emphasized the necessity of fluid replacement, so that all major operations now are done with an intravenous needle in place through which fluid is introduced during the time of the operation and immediately afterward, and, if necessary, any blood lost can be immediately replaced by a transfusion. By these precautions the incidence of postoperative shock has been reduced enormously and blood pressure readings can be kept at satisfactory levels throughout the operative procedure and the immediate postoperative period.

The fourth landmark in the progress of urology in the last 25 years has been the development of endocrine methods in the management of carcinoma of the prostate. It has been well known for many years that castration would result in atrophy of the prostate gland, and indeed this procedure was advocated many years ago in the treatment of benign prostatic hypertrophy. On account of the uncertain results this procedure was soon discarded.

The influence of hormones on the progress of prostatic cancer was first studied and definitely demonstrated by Huggins, who showed that in most patients the progress of the disease could be greatly inhibited by complete removal of the

androgen factor by castration, and by Herbst, who showed that in many patients a similar although temporary inhibition could be obtained by the administration of estrogens by which the androgen secretion of the testis could be inhibited. It undoubtedly is true that by the use of these methods malignant disease will undergo varying degrees of regression. In some patients even this process is so marked that several months after the institution of either treatment a diagnosis of prostatic cancer could not be made on rectal examination. This regression has been explained by the fact that actively growing neoplastic cells at the periphery of the growth require more androgens for their development and thus undergo atrophy and necrosis. However, unfortunately at varying periods of time the endocrine effect disappears and the cancer cells again proliferate and extend. It, therefore, is obvious that although endocrine therapy may result in marked regression of the neoplasm, complete destruction of all cancer cells has not been obtained, and in fact no case has yet been reported in which a complete eradication of cancer of the prostate has been obtained by endocrine means alone. Therefore, to attain the surgical ideal in dealing with all types of cancer the only method available to us at present is the radical operation.

With the knowledge that endocrine therapy will result, in many cases, in marked regression of the neoplasm, it must be assumed that this regression means the destruction of many of the malignant cells. On account of this fact it has been our custom to administer estrogens to all patients in whom the radical operation is considered, even though some of them from clinical examination may apparently be in a very early stage. If we have an agent which is capable of destroying at least some of the malignant cells it seems to me that this agent should be employed before surgical methods are undertaken, with the hope that the more actively growing neoplastic cells will be destroyed and therefore the ultimate prognosis for complete eradication will be enhanced. Surprising regression often may be obtained in patients who seem on first examination to have cancer far too extensive for the radical operation. In some of these individuals the neoplasm has regressed to such an extent that it was considered that the radical operation would offer a good chance of complete eradication. We have had a short series of such cases in patients observed about 10 years ago on whom following regression from endocrine therapy, the radical operation was done. One of these patients is alive at present with no clinical evidence of recurrence or metastasis eight years after the operation—an individual in whom at first examination the neoplasm had been considered far too advanced for successful removal but, under estrogen therapy, there was such regression of the neoplasm that radical operation was considered feasible and this procedure was done successfully. It also has been our custom, therefore, to administer estrogens for certain periods following the radical operation in the hope that any cells which may have been beyond the scope of the operation might be destroyed by this treatment. Naturally, however, figures to substantiate this hypothesis would be extremely difficult to obtain.

Many studies have been made on the survival rate following the radical operation and most observers are in agreement that approximately 50 per cent of the

patients subjected to radical prostatectomy have survived five years or more. Jewett has found that the 10 year survival rate was 28 per cent. He further studied a series of postoperative specimens and found that in the group in which no extension of the neoplasm into the capsule was demonstrable the 10 year survival rate was 49 per cent—only slightly less than the expected survival rate in individuals of this age.

As has been mentioned before, after varying periods of time, the neoplasm which may have regressed to a marked degree as a result of endocrine therapy, again begins its progress and estrogens obviously have no further effect on its extension. It has been assumed that this regrowth might be due to androgen stimulation from the adrenal gland. Therefore, adrenalectomy was proposed some years ago and done in a number of patients. However, this operation now has largely been discarded because, except for the occasional patient in whom a temporary relief of symptoms was afforded, the results have been disappointing. W. J. Baker made a careful follow-up study on a series of his own cases and has concluded that "After bilateral adrenalectomy in patients with advanced cancer of the prostate gland serial x-rays of osseous lesions, serial acid and alkaline phosphatase determinations, serial rectal examinations, serial body weight and hemoglobin determinations and serial androgen values have furnished us no evidence that prostatic cancer or its metastases have been inhibited. As far as we can ascertain, the cancer continues to grow at its usual rate."

The use of cortisone recently has been proposed to produce the so-called *medical adrenalectomy*. With this therapy improvement in the local lesion may occur and a beneficial clinical effect also is often noticeable, so that in relapsed cases of extensive carcinoma of the prostate it has been our plan to institute cortisone therapy, but these patients should be carefully controlled to avoid toxic effects.

Great interest has been aroused in the past few years by the use of radioactive isotopes in the treatment of extensive prostatic cancer. Flocks has treated a series of such patients with interstitial injection of radioactive gold and Rusché has used radioactive colloidal chromic phosphate. The ultimate results of these agents will not be known for some years. Unless great precautions are taken, damage to all those engaged in this work is possible. Severe complications have been reported in some of the patients treated. Edgar Burns who has used radioactive gold has summed up his impression as follows: "Gold is not a substitute for x-ray or surgery. So far, it has not been curative in any instance. It is merely palliative and its use is still primitive and experimental. Laboratory experiments and human trial indicate that, with further improvement in the techniques of administration and dosage, it will become a valuable adjunct in the treatment of advanced cancer."

SUMMARY

During the past 25 years we have witnessed an epoch making advance in the whole field of medicine. In the specialty of urology this progress has nowhere been more outstanding, and the result of these advances has been a steady im-

provement in our methods of diagnosis and a steady reduction in morbidity and mortality rates, and consequently these facts have resulted in great relief of human suffering. The perfection of excretion urography, with the introduction of newer chemicals which have been progressively safer with the virtual elimination of all serious reactions, has been a great contribution toward the perfection of diagnosis of all diseases of the genitourinary tract. The introduction of sulfonamides and the antibiotics has witnessed a similar evolution in that more effective products are continually being offered by research laboratories, which provide a broader spectrum of effect on organisms and a notable diminution of toxic effects.

In the field of surgical technic these 25 years have witnessed the perfection of the operation of transurethral resection and its proper evaluation. A further epoch making advance has been the development of endocrine methods in the management of carcinoma of the prostate, and with our continued understanding of endocrine effects on malignant disease great hope can be held out for the future.

Let us hope, therefore, that the succeeding 25 years will witness, as I am sure it will, similar advances in the diagnosis and management of diseases of the genito urinary tract.

ROUTINE SUPRACLAVICULAR BIOPSY IN SUSPECTED BRONCHIOGENIC CARCINOMA

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We believe that biopsy of the supraclavicular nodes is helpful often enough to be used frequently *or even routinely* in all cases of suspected bronchiogenic carcinoma before major surgery is done. It is one more device we may use to help us answer the question 'Will a major operation actually help this particular patient'? Papers on the general topic of bronchiogenic carcinoma usually list under diagnostic procedures the following: bronchoscopy, bronchograms, needle biopsy and exploratory thoracotomy. Only rarely is supraclavicular biopsy of lymph nodes mentioned.

HISTORIC AND PHILOSOPHIC CONSIDERATIONS

In 1942 Ochsner and DeBakey[13] collected the incidence of lymph node metastases in 1298 cases of carcinoma of the lung. In their series only 4.2 per cent of the supraclavicular nodes were involved. The highest incidence was in the tracheobronchial nodes in which there was involvement of 69.7 per cent. However, cervical nodes were listed as involved in 17.4 per cent, and it is probable that the cervical and supraclavicular nodes should be added together because both can be reached by the supraclavicular approach. Axillary nodes were involved in 6.6 per cent in their series and femoral-inguinal nodes in 2.2 per cent. Submaxillary nodes were involved in 3 per cent. The other lymphatic involvement was in relatively inaccessible locations for minor surgery.

William Reinhoff[17] in 1950 analyzed 344 cases of bronchiogenic carcinoma that were inoperable. In addition to metastases in the regional lymph nodes, the sites of metastases in the order of frequency of their involvement were (1) supraclavicular and (2) axillary lymph nodes.

Kikuth[9] believed that the regional lymph nodes (tracheobronchial nodes) are involved in practically all cases. Koletsky[11] noted in 88 cases that these nodes were involved in 82.9 per cent. Olson[15] observed regional lymph node involvement in 97 per cent in his series of 67 cases. Ochsner and associates[14] reported 3,047 collected cases in which the regional tracheobronchial lymph nodes were involved in 72.2 per cent. Camblos and co-workers[3] noted peripheral lymph nodes (axillary, supraclavicular, etc.) positive in 13.9 per cent of their 115 cases.

Craver[5] analyzed 175 cases of primary carcinoma of the lung and noted the methods of making the diagnosis in preparation for treatment. Incision or aspiration biopsy of lymph nodes made the diagnosis in 7 cases and added confirmation by this means in 5 other cases. Other methods used in his series were tho-

racotomy, examination of expectorated tissue, bronchoscopy, aspiration biopsy of the lung, aspiration biopsy of ribs, and a group of 15 cases in which no diagnostic tissue could be obtained during life, but subsequently was confirmed by autopsy.

King[10] stated that in 15 per cent of their cases a correct diagnosis was made by biopsy of cervical or axillary lymph nodes. He recommended that a very careful examination of the neck and axillae be made in all suspected cases.

Carr and associates[4] mentioned that the "physical examination should include a particularly careful search for enlarged lymph nodes in the supraclavicular fossae, axillae and abdomen. In fact the entire body should be palpated for nodules. A recent patient in our Clinic was spared an exploratory operation by the discovery of a small pea-sized subcutaneous metastatic node on the anterior chest wall."

Ornstein and Epstein[16] analyzed 26 autopsy cases at Seaview Hospital in patients with primary bronchial carcinoma. They found no involvement of the cervical nodes and only 2 cases with axillary involvement. The most common involvement was in the tracheal and bronchial nodes, noted in 15 cases each. The mediastinal nodes were involved in 5 and abdominal nodes in 1.

Beeler and Irey[1] reported a clinico-pathologic study of 50 autopsied cases of bronchiogenic carcinoma. Bronchoscopy was done in 11 cases with positive findings in 6. Peripheral lymph node biopsy confirmed the clinical diagnosis six times and was negative once.

Lindskog[12] reported on 100 primary pulmonary cancers; the diagnosis was made by lymph node biopsy in 10 of these patients. Other methods by which the diagnosis was made included bronchoscopy (28 patients) thoracotomy and biopsy (10 patients) resection of lung (10 patients), sedimentation of pleural fluid (5 patients) needling of lung (5 patients) thoracoscopy (2 patients) and biopsy of metastases (7 patients). Metastases were found in the central nervous system (2 patients), skin (1 patient), thyroid (1 patient) and bone (2 patients).

Storey and Reynolds[19] recently have reemphasized the importance of resection of the deep cervical fat pad and its nodes, as well as mediastinal biopsy and lung biopsy through an open thoracotomy. They suggested that the usefulness of these technics has not been fully appreciated. They stated that "The concept that intelligent treatment demands an accurate diagnosis is universally accepted by the profession. It has been only in comparatively recent years, however, that this fundamental principle has been fully applied to intrathoracic lesions. In the past the policy of watchful waiting frequently was adopted; therapy was applied on the basis of a presumptive diagnosis, or trial and error methods were employed. If metastatic malignancy, compatible with a primary pulmonary lesion, is found in the deep cervical or superior mediastinal nodes upon resection of the deep cervical fat pad and its contained nodes, the case usually is judged inoperable". Storey and Reynolds do not always adopt this policy. They frequently still use palliative resections when there is known widespread mediastinal metastases, but they admit that "careful follow-up studies may well show that the degree of palliation received by pulmonary resection when the nodes in

the deep cervical fat pad are involved, is not, in the usual case, sufficiently worth-while to justify pneumonectomy. In any event the resectability rate in such conditions is low. It certainly would seem that in poor-risk patients where the question of resectability is great, proved metastases in the deep cervical, subclavicular, or superior mediastinal nodes would weigh heavily against the decision to attempt extirpative surgery".

Brindley and Miller,[2] in 600 lymph node biopsies in a variety of diseases, noted that the cervical nodes showed the highest incidence of positive results, while the inguinal nodes gave the lowest results. Tuberculosis was diagnosed in about one-eighth of their cervical biopsies and they conclude that a definite diagnosis can be established by lymph node biopsy in approximately 56 per cent of the lymph node specimens received at the pathology laboratory. Their series included 40 cases of lymphosarcoma, about half of which were diagnosed from cervical node biopsy.

Daniels[7] presented 5 cases. In none of his cases were the nodes palpable before operation. Boeck's sarcoid was diagnosed in the first 2 cases, metastatic carcinoma in the third, all of which prevented major surgical explorations of the chest. In the fourth case a major exploration was done only to find inoperable carcinoma of the lung with metastases. A supraclavicular biopsy was then done and 3 small nodes were found which all contained metastatic carcinoma. If the biopsy had been done first, the exploratory thoracotomy would not have been necessary. Interestingly enough, the fifth case demonstrated silicotic changes in the nodules and silica particles could be seen by using Nickol prisms. The last case illustrates the fact that silicosis, as well as sarcoidosis and tumors, advance as far as this group of nodes.

The remarks of Harkins[8] in a discussion of Cotton and Penido's paper on pleural pulmonary resection are apropos. He said that "I should like to confine myself to this section of this paper that involves what we, in our clinic, would regard as meddlesome surgery for obviously hopelessly inoperable carcinoma of the lung. Our tendency has been to do less and less rather than more and more; in other words, to try to make a cleaner, clearer distinction between inoperability and operability, and to work toward a more specified procedure consistent with a cancer operation. We have explored the neck and removed the lymph nodes routinely in an effort to establish inoperability before proceeding with surgery. This neck dissection also is useful as a diagnostic maneuver in some instances where a diagnosis of cancer cannot otherwise be made. In such a situation it not only establishes the diagnosis but the inoperability thereof. We have, I believe by this dissection of the neck spared some 50 per cent of our patients surgery that was otherwise unproductive. We can be guilty of errors of commission as well as errors of omission. The excision of malignant tumors is not always palliative."

A recent paper by Shefts and associates[18] present some very pertinent findings. The authors believe that a simple scalene node biopsy is comparable to a biopsy of the mediastinal nodes without the necessity of entering the thorax. The percentage of positive findings in the nodes varies with the pathologic conditions

and the accuracy of the dissection. These writers have purposely refrained from going down into the mediastinum in an effort to make the operation as simple and safe as possible. In 2 of their cases nodes were reported as negative and re-operation on the same side resulted in a positive result in both cases, (Boeck's sarcoid). It is important to obtain the proper scalene nodes, for in 3 cases in their series nodes just superficial to the deep fascia were normal although those immediately deep to the fascia revealed positive findings.

Five other patients in whom nodes were not found at operation refused re-operation. Their series consisted of 205 scalene node dissections, including 17 bilateral dissections in a total of 187 patients. Unfortunately, it was only in the latter part of their series that routine cultures were made from the nodes for tuberculosis and fungi. This is an extremely important part of the examination and it is believed by them that the chance of the proper diagnosis in many cases was almost certainly missed because of failure to obtain these cultures.

Complications mainly were limited to involvement of the thoracic duct. The left thoracic duct was torn once and the right twice, a total of three complications in 205 dissections (1.4 per cent); in none of these was the complication fatal.

These authors were very strict in their interpretation of their percentages and *only those cases not diagnosed by other methods* were considered in their percentages. In the entire series of 187 patients there were 67 patients (35.8 per cent) in whom the biopsy material revealed positive evidence of the identity of the intra-thoracic disease which previously had been undiagnosed despite application of the generally used nonsurgical diagnostic measures. They did 136 right-sided biopsy dissections in contrast to only 69 on the left, believing that in instances of equal bilateral thoracic involvement, the right scalene biopsy was the safer procedure. However, usually they selected the scalene nodes on the same side as the principal intrathoracic involvement whenever there was a choice. They did 17 bilateral neck dissections and 9 nodes showed positive findings. There were only two instances in which both the right and the left scalene nodes were positive. They suggest that if the node biopsy on one side is negative, it might be advisable to dissect the other side. Boeck's sarcoid was by far the most common diagnosis in their series of 187 patients. Other disease processes found in the scalene nodes included bronchiogenic carcinoma, tuberculosis, lympho-sarcoma, Hodgkin's disease, fungus infections and metastatic carcinoma from the lungs. In all of their cases in which the scalene nodes were positive for tuber-culosis, the nodes were enlarged, grossly necrotic and positive for tubercle bacilli by tissue stain and by culture. In the lymphomas the microscopic node diagnosis may not only eliminate an unnecessary thoracotomy but also lead to the prompt use of presently accepted therapy. In one of their cases diagnosis was made by culture of the scalene nodes which yielded histoplasma.

Shefts and associates summarize by saying that scalene node biopsy should be done whenever it is advisable to know the contents of the mediastinal nodes. It is of value in all cases of bronchiogenic carcinoma to determine whether or not the carcinoma has extended beyond the immediate confines of the chest. It

should be done in all cases of suspected bronchiogenic carcinoma. This actually
will include all roentgenographic opacities not otherwise identified, especially
the small isolated, often peripheral, asymptomatic lesions. Scalene node dissec-
tions should be of value in cases of medico-legal interest (pneumoconiosis)
wherein the offending foreign body substances may be identified.

From August 1949 to February 1953 prescalene lymph node biopsies were
done in 41 patients at the Veterans Administration Hospital, Fort Logan and
Denver. Cuykendall reported[6] that a bronchoscopic examination was made in
every case. Bronchial washings, sputum, gastric washings and pleural fluid were
studied bacteriologically and cytologically. When the possibility of involvement
of the lung by neoplasm or granuloma remained, prescalene lymph node bearing
tissue was removed in an attempt to show metastases or involvement by granu-
loma. Specific disease processes were found in 8 cases in the prescalene lymph
nodes. In 3 cases metastatic carcinoma was present; in 4 cases noncaseating
tuberculoid granuloma (Boeck's sarcoid) was discovered and in 1 case a lympho-
sarcoma was found. In 1 of the cases of noncaseating tuberculoma, biopsy ma-
terial taken from the contralateral side after an absence of 6 months again
showed the granuloma. In another of the cases of noncaseating tuberculoid
granuloma, a biopsy in which an inadequate quantity of material was obtained
was followed 10 days later by an adequate biopsy. In 41 cases in which pre-
scalene biopsy was done, there were 14 cases of bronchiogenic carcinoma; in 3
of these prescalene lymph node biopsies showed metastases to the lymph nodes.
Four cases of noncaseating tuberculoid granuloma (Boeck's sarcoid) were seen;
in all 4 cases biopsy yielded positive results. Biopsy of prescalene lymph nodes
in cases in which palpable lymph nodes were absent has proved to be of value
in the diagnosis of intrathoracic diseases, especially the granulomas, (Boeck's
sarcoid) and malignant neoplasms (table I).

TECHNIC OF REMOVAL OF SUPRACLAVICULAR NODES

The operative approach differs in no important respect from that used for
temporary paralysis of the phrenic nerve. A $1\frac{1}{2}$ or 2 inch incision is made above
the clavicle, centering over the posterior border of the sterno-cleido-mastoid
muscle and about $\frac{1}{2}$ inch above the clavicle. The skin, subcutaneous tissues and
platysma, and the superficial layer of the deep cervical fascia are divided, bring-
ing the deep cervical fat pad into view. The fat pad is excised with all contained
nodes down to the anterior scalene muscle. Medially the resection extends to
the carotid sheath, posteriorly to the middle scalene muscle, and inferiorly down
to the subclavian vein. If insufficient tissue then is obtained, the resection may
then be extended down into the superior mediastinum along the great vessels.
By traction on the fat pad from above a large mass of adipose tissue can be de-
livered into the neck from the subclavicular and retrosternal region. This mass
of fat invariably contains nodes which may be enlarged and easily recognized
or they may be so small that they can be detected only after careful search by a
pathologist. The operation causes minimal morbidity and no mortality.

Daniels[7] suggested that in men the lateral aspect of the clavicular portion of

TABLE I

Lymph node metastases in bronchiogenic carcinoma

Author	Year	Number Cases	Supraclavicular	Tracheo Bronchial	Axillary	Femoro-Inguinal
Ochsner and DeBakey	1942	1298 (collected)	4.2% supraclavicular plus 17.4% cervical plus 3% submaxillary for a total of 24.6%	69.7%	6.6%	2.2%
Reinhoff	1950	344, All inoperable	Second most common	Most common	Third most common	
Kikuth	1925	?		In practically all cases		
Koletsky	1938	88		82.9%		
Olson	1935	67		97%		
Ochsner	1945	3047 (collected)		72.2%		
Camblos	1949	115	13.9% peripheral node involvement (axillary, supraclavicular, etc.)			
Craver	1940	175	Incision or aspiration biopsy of lymph nodes made diagnosis in 4% of their cases and added confirmation in 3% in other cases			
King	1938	72	15% of cases diagnosed by biopsy of cervical or axillary nodes			
Ornstein	1941	26 autopsy cases		57.7% plus 19.2% mediastinal nodes	7.7%	
Beeler	1950	50 autopsy cases	Peripheral lymph node biopsy confirmed diagnosis in 12% of cases and was negative in 2%			
Lindskog	1946	100	10% diagnosed by peripheral lymph node biopsy and another 7% by biopsy of metastases			
Brindley and Miller	1950	600 cases with a variety of diseases	The cervical nodes gave the highest incidence of positive results, and the inguinal nodes the lowest results. 56% of the various nodes submitted gave a definite diagnosis			
Daniels	1949	5 cases with various diseases	In 100% of their cases the nodes gave material for a definite diagnosis			
Shefts	1953	187 cases, 17 bilateral, a total of 205 dissections	35.8% (67 cases) diagnosed by the biopsy alone—in many others, the biopsy was positive confirming the already established diagnosis			
Cuykendall	1953	41 cases	19% (8 cases) diagnosed by supraclavicular biopsy			
Skinner and associates	1955	100 cases	43% yielded positive diagnoses by supraclavicular biopsies			

SUMMARY OF ABOVE TABLE

In bronchiogenic carcinoma, we can expect the supraclavicular nodes to contain typical carcinomatous tissue in from 20 to 40 per cent of all cases. Obviously, the more advanced or immature the carcinoma, the higher the percentage of positive results will be.

the sternocleidomastoid muscle may be divided, although this may not be necessary in women. We have not found it necessary to cut the muscle in any of our patients, merely splitting it or retracting it with phrenic retractors. The floor of the space upon which the fat pad is found is formed by the anterior scalene muscle with the phrenic nerve lying in its sheath. The transverse cervical and inferior thyroid vessels usually are encountered coursing through the fat, and usually can be retracted without difficulty. If injured they may require ligation. Weiss[20] stated that the supraclavicular fat pad contains lymphoid tissue which is part of the mediastinal drainage system. If the lesion is in the left upper lobe then a left-sided approach is used. In all other cases we usually prefer to explore the right side of the neck because: (1) All of the right lung and the left lower lobe and the lower portion of the left upper lobe drain into the right supraclavicular nodes, and (2) the thoracic duct is smaller on the right side and there is less danger of damaging it in the resection (Fig. 1).
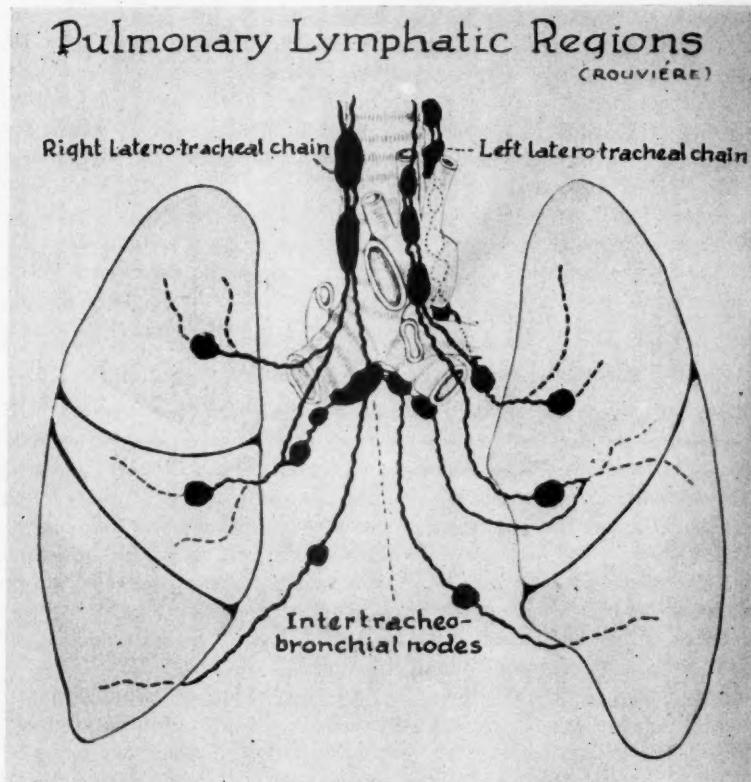
FIG. 1. Pulmonary lymphatic regions after Rouviere

TABLE II

Age	Number of Patients
0–10	3
11–20	3
21–30	4
31–40	12
41–50	14
51–60	26
61–70	27
71–80	11

Statistical Analysis of 100 Supraclavicular Biopsies

In our series of 100 consecutive supraclavicular biopsies, 70 were in male patients and 30 in female patients. The average age of all patients was 53 years, the youngest being 4 years of age and the eldest 78 years of age (Table II).

Only 1 patient with a supraclavicular biopsy positive for cancer had the chest explored. Two other patients with positive biopsies had resections of neck tumors. *Positive biopsy reports were obtained in 43 of the 100 patients.* Forty-one of the 100 patients had a negative supraclavicular biopsy report and 16 other patients had negative biopsies when positive biopsies could not reasonably be expected; they had a medical disease such as bronchiectasis or pneumonia. If we add the negative biopsies of the the 16 medical cases *we had a total negative biopsy incidence of 57 per cent, and 43 per cent positive reports.*

In 10 patients the diagnosis was made clinically of bronchiogenic carcinoma but was not proved by tissue study. None of these patients had a positive supraclavicular biopsy.

In 4 patients no tissue was sent to the laboratory for examination as the surgeon did not believe that clinically important nodes were present. This was an error as sometimes small nodes contain microscopic cancer. We now believe that tissue should be sent to the laboratory in every case, both for permanent tissue section and in many cases for culture for acid fast organisms, fungi and pyogens.

In 18 patients no nodes were palpable and yet supraclavicular biopsy yielded a positive report. In 25 patients the nodes were palpable and the supraclavicular biopsy was positive. Cultures were made on only a few of the nodes in our series. We are now culturing many more of these nodes and hope to improve our yield of positive results.

Our patients had numerous other diagnostic procedures without establishing a diagnosis before the supraclavicular biopsy was done. These studies included skin tests for tuberculosis and histoplasmosis, bronchoscopy and bronchial washings, bronchograms, multiple sputum studies for tubercle bacilli, tumor cells and fungi, pleural fluid studies when fluid was present, bone marrow examinations, fluoroscopy, roentgenograms of the chest in various projections and

even, in a few cases, lung biopsy or biopsy of the chest wall and axillary nodes.

Resection of the right or left supraclavicular nodes was almost equal in our series of 100 cases. No case had a bilateral neck biopsy.

Three patients had combined tuberculosis and bronchiogenic carcinoma. All 3 received nitrogen mustard therapy and 1 in addition received deep roentgenologic therapy. The roentgenologic treatment in this case caused no difficulty, although we sometimes believe that such deep therapy is contraindicated in active pulmonary tuberculosis.

Nine of the patients with negative supraclavicular biopsies were explored, but not resected as the cancer was found to be incurable surgically. Two patients refused exploration. Of the resections in patients with negative supraclavicular biopsies there were 6 pneumonectomies for bronchiogenic carcinoma and 6 lobectomies, 1 of which was a bilobectomy. These lobectomies were done for carcinoma in poor-risk patients, and for lung abscess, tuberculosis and chronic pneumonitis. The cancer patients who do not have resections usually have therapy consisting of deep roentgenologic treatments or nitrogen mustard or tri-ethylene melamine or some combination of these treatments. One patient was especially interesting; 5 previous biopsies of a chest wall tumor and axillary nodes were not diagnostic. A supraclavicular biopsy showed Hodgkin's disease.

In the group of 43 patients with positive biopsy reports in this series, 41 had no definite diagnosis prior to the positive supraclavicular biopsy report. These patients had had many other clinical tests. In 1 case, several biopsies of a chest wall tumor had been done previously without a definite diagnosis being established. The other 2 patients with positive supraclavicular biopsy reports already had the type of their disease known as these were cancers primary elsewhere in the body with metastases to the lung. Thus, in 41 of the 100 patients the first positive cellular diagnosis was established by the supraclavicular biopsy (table III).

CASE REPORT

Mr. D. D. was a 49 year old white man. In October 1953 he began having arthritis in his knees and ankles with some ankle edema. In November 1953 he had a cold with high fever with pleurisy and unproductive cough. (He had been a chain smoker for the past 10 years.) In December 1953 chest roentgenograms showed a mass in the upper lobe periphery of the left lung 8 cm. in diameter. Cortisone was started for his arthritis, without much effect.

The physical examination was noncontributory, except for an enlarged soft node above each clavicle and osteoarthropathy of moderate degree, with slight pitting edema of the ankles.

He was admitted to the Baptist Memorial Hospital on Dec. 30, 1953 for further studies. A gastrointestinal roentgenographic study showed a filling defect in the greater curvature of the stomach which was thought to be a primary gastric neoplasm. However, at exploratory laparotomy no neoplasm was found. Bronchoscopy was negative as were also cell studies.

On Jan. 18, 1954 a left supraclavicular exploration was done and several large nodes were removed from the superior mediastinal chain. These were interpreted on microscopic section as "Metastatic undifferentiated carcinoma to lymph node".

Palliative resection of the lung tumor was decided against, as the patient was relatively comfortable, and a course of nitrogen mustard therapy was recommended. He was discharged from the hospital on Jan. 20, 1954.

TABLE III

Disease	No. Cases	Positive Supra-Clavicular Biopsy	Negative Supra-Clavicular Biopsy
1. Unclassified Bronchiogenic Carcinoma	17	0	17
2. Epidermoid Bronchiogenic Carcinoma	5	2	3
3. Undifferentiated Bronchiogenic Carcinoma	24	16	8
4. Adenocarcinoma	7	6	1
Bronchiogenic Carcinoma, Total	53	24	29*
Metastatic Carcinoma to lung	11	5	6
Neuroblastoma	2	2	0
Neurilemoma	1	1	0
Hodgkin's Disease	3	3	0
Lymphoma	1	0	1
Lipoma	1	1	0
Undiagnosed Hilar Mass	1	0	1
Carcinoma of Esophagus	1	0	1
Pulmonary Adenomatosis	1	0	1
Boeck's Sarcoid	5	5	0
Pulmonary Tuberculosis	2	0	2
Streptobacillus Infection	1	1	0
Chronic Mediastinitis	1	0	1
Bronchiectasis	4	0	4
Lung Abscess	1	0	1
Pneumonia	4	0	4
Pulmonary Infarct	1	0	1
Histoplasmosis	1	0	1
Idiopathic Pulmonary Fibrosis	2	0	2
Cardiac failure with Jugular Vein thrombosis	1	0	1
Congestive heart failure with Pleural effusion	1	0	1
Colloid Goitre, adenomatous	1	1	0
Total	100	43	57

*Ten of these diagnoses were not proved by tissue examination. No positive biopsies are included in this group. These patients were considered inoperable because of clinical signs.

Comment. Roentgenograms showed "mass in the lung and another in the stomach". Laparotomy revealed a normal stomach. If the supraclavicular biopsy had been done first, the patient would have been spared the discomfort and expense of a needless exploratory laparotomy.

SUMMARY AND CONCLUSIONS

A series of 100 patients suspected of having bronchiogenic carcinoma were subjected to supraclavicular biopsy.

A statistical analysis of 100 consecutive supraclavicular biopsies is presented. In many of these patients a positive biopsy saved the patient major surgery, as such distant metastases made surgical cure highly improbable.

Supraclavicular biopsy was performed irrespective of whether or not nodes were palpable.

In 18 patients no nodes were palpable and yet supraclavicular biopsy yielded a positive report.

In all patients subjected to supraclavicular biopsy the prescalene fat pad and the nodes therein should be sent to the laboratory for pathologic study and also for bacteriologic examinations.

In 100 consecutive supraclavicular biopsies, 43 positive reports were obtained which we believe is a highly profitable yield since even lung biopsy is not always 100 per cent diagnostic.

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PSEUDOCYST OF THE PANCREAS ASSOCIATED WITH HYDROTHORAX

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The occurrence of pleural effusion secondary to pancreatitis is not unusual, but hydrothorax associated with pseudocyst of the pancreas is rare. The presence of hydrothorax in a patient with an upper abdominal mass often suggests malignant disease, and operation might be deferred if no diagnosis is considered except a metastatic malignant process.

In 1953 Gross and co-workers[7] collected from the literature 5 cases of pseudocyst of the pancreas associated with hydrothorax and added 1 case of their own. It is the purpose of this paper to add 1 personal case and to include 4 others, bringing to 11 the total number of reported cases of pseudocyst of the pancreas associated with hydrothorax (table 1).

REPORT OF A CASE

C. B., a white man, candy and newspaper vendor, 52 years of age, was admitted to the Jefferson Davis Hospital, Houston, Texas, on March 17, 1953, because of weakness of one month's duration. He had had progressive swelling and tightness of the abdomen, as well as progressively severe dyspnea, during the week prior to admission, but no nausea or vomiting had occurred. Since the onset of his present illness, he had been constipated and had taken laxatives frequently. His stools were small but otherwise were normal. During the preceding two to three months his appetite had been poor, and he had lost 25 pounds in weight. From 1935 to 1950, he had consumed considerable quantities of alcoholic beverages but had drunk little since then. There was no history of jaundice.

Physical examination showed a well developed white man who appeared to be acutely and chronically ill, with evidence of recent weight loss. His temperature was 98.4 F., and his pulse rate was 120 per minute. There was splinting of the right side of the chest with intercostal retraction. Flatness to percussion was present on the right side, below the midscapular area posteriorly, and anteriorly below the second rib, with decreased breath sounds and vocal and tactile fremitus. The left lung was clear. There was abundant abdominal ascites with a fluid wave and flank dullness. No masses were palpable. The remainder of the examination was not remarkable.

The hemoglobin was reported to be 17.7 Gm. per 100 cc., and the white blood count was 42,800 cells per cu. mm., with a pronounced shift to the left. The urinary specific gravity was 1.024. Except for one-plus albuminuria and a fair number of bacteria, the urine was normal. The serologic test for syphilis was negative.

A roentgenogram of the chest on March 17 (fig. 1) showed pleural effusion obscuring the lower two-thirds of the right pulmonary field. A roentgenogram of the abdomen (fig. 2) demonstrated a well-defined, homogeneous density 16 cm. in diameter filling the middle and left portions of the upper abdomen and depressing the transverse colon. Scattered amorphous calcification was seen within the density just lateral to the transverse process of the second lumbar vertebra on the right in the region of the head of the pancreas. On the same day right thoracentesis was done (table 2) with removal of 1950 cc. of lime-colored, slightly

TABLE 1

Pseudocyst of the pancreas associated with hydrothorax—clinical data

Collected and Personal Cases

Author	Year of report	Color, Sex	Age (yrs.)	Diagnosis	Pleural Effusion		
					Side	Studies for enzymes	Explanation
Phillips[13]	1901	M	22	Traumatic pseudocyst of pancreas with pleural effusion	Left	Not done	Not given
Alivisatos[1]	1920	WF	26	Polycystic pancreas with pleural effusion (probably chronic pancreatitis)	Right	Not done	Small right diaphragmatic hiatus just above crus, allowing thoracic and abdominal cavities to communicate (autopsy)
Jones[19]	1944	WF	49	Degenerative cyst of pancreas due to pancreatic necrosis, with extension into mediastinum through esophageal hiatus and rupture into right pleural cavity	Right	Not done	Extension of cyst into mediastinum through esophageal hiatus with rupture of cyst into right pleural cavity
Traquair[16]	1946	WM	23	Traumatic pseudocyst of pancreas; after drainage, a chylous left pleural effusion developed	Left	Not done	Multiple intraabdominal injuries, pancreas, biliary apparatus, lymphatic vessels; granulating tissue in left subphrenic space
Schieppatti[14]	1946	WM	50	Pseudocyst, postpancreatitic	Left	No enzymes present	Exudate of pancreatitis drained toward left diaphragm; subsequent pleural effusion result of direct diffusion
Bickford[2]	1948	WM	20	Traumatic pseudocyst of pancreas, with injury of thoracic wall, multiple fractures, and left hemothorax	Left	Not done	Result of injury to thoracic wall or lung at time of initial accident
Bickford[2]	1948	WM	21	Traumatic pseudocyst of pancreas; ruptured spleen	Left	Not done	Result of injury to thoracic wall or lung at time of initial accident
Edlin[5]	1951	NM	60	Chronic recurrent pancreatitis, with pseudocyst of pancreas extending through esophageal hiatus into mediastinum	Left	Not done	Possibly due to mild degree of cardiac decompensation present or to the pancreatic disease that was probably smoldering under left diaphragm

				...pseudocyst of pancreas or to the pancreatic disease that was probably smoldering under left diaphragm			
				...through esophageal hiatus into mediastinum			
Gross, Null, Loeb[7]	1952	NM	27	Pseudocyst of pancreas, due to pancreatitis with pleural effusion	Not done	Left	(1) Passage of an irritative substance through diaphragmatic lymphatic vessels to pleural cavity, where an irritative pleural effusion was set up, or (2) transperitoneal passage of ascitic fluid into the pleural cavity through physiologic diaphragmatic openings
Smith[15]	1953	NM	44	Recurrent pseudocyst of pancreas, due to pancreatitis with hemorrhagic pleural effusion	Not done	Right	Not given
Mahaffey[22]	1954	WM	52	Pseudocyst of pancreas, due to pancreatitis with pleural effusion	With few exceptions, amylase levels in pleural effusion higher than in serum	Right	Probably result of diffusion of inflammatory edema from subphrenic space, aided to some extent by decreasing pressure gradient from abdomen to thorax and action of diaphragm
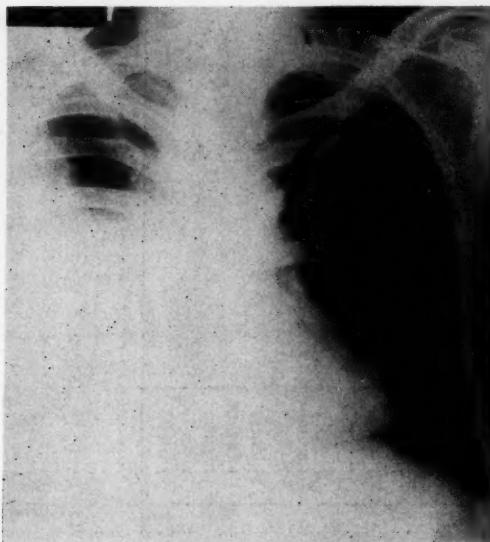
FIG. 1. Upright posteroanterior film of chest, taken on admission, showing extensive right pleural effusion.

turbid fluid with a specific gravity of 1.016 and total protein of 4.3 Gm. per 100 cc. The culture was negative. Cell block of the pleural fluid subsequently was reported as suspicious of malignant disease; there was a predominance of rather large, apparently mesothelial cells, many occurring in varying sized lumps.

On March 18, the following liver studies were obtained: bromsulphalein liver excretion test, 26 per cent dye retention at the end of 45 minutes; serum bilirubin, 0.2 mg. per cent; thymol turbidity, 3 units; total serum protein, 5.2 Gm. per 100 cc., albumin 3.3 Gm. per 100 cc., globulin 1.9 Gm. per 100 cc., and alkaline phosphatase 3.9 Bodansky units.

Thirty-one hundred cu. cm. of light-green, thin fluid was removed by abdominal paracentesis on March 19 (table 2). Thoracentesis yielded 950 cc. of straw-colored fluid. A firm, slightly tender ballottable mass, which moved with respirations, was noted in the left upper abdominal quadrant for the first time on March 23. On March 26 a barium enema (fig. 3) showed the mass to be extrinsic to the colon, depressing the transverse colon, and occupying the left upper abdominal quadrant and epigastrium. Upper gastrointestinal roentgenographic studies (figs. 4 and 5) on March 30 showed a large mass extrinsic to the colon, arising posteriorly, displacing the antral region anteriorly and slightly inferiorly. Bronchoscopy did not disclose any evidence of bronchial neoplasm to explain the pleural effusion.

On April 6, at exploratory laparotomy, approximately 2500 to 3000 cc. of cloudy, turbid, ascitic fluid was removed. The peritoneum generally was inflamed, reddened, and thickened, but no purulent exudate was noted. There were fibrinous adhesions in the right subdiaphragmatic and subhepatic spaces and about the foramen of Winslow which was sealed. The liver otherwise was normal. No openings were palpable in the right diaphragm. The gallbladder was normal. A few areas of fatty necrosis were noted in the mesentery of the small intestine. The spleen was enlarged to one and one-half times its normal size but otherwise was normal. The stomach was displaced anteriorly and was compressed by a large cystic mass in the gastrocolic and transverse mesocolic areas. The cyst was incised, and approximately 3000 cc. of clear tea-colored fluid was removed. The wall of the cyst was smooth and thick. The head of the pancreas was enlarged and firm, and an area of calcification could be pal-

FIG. 2. Flat plate of abdomen. Large midabdominal mass with amorphous calcification is well seen.

pated within the head of the pancreas. There was no evidence to suggest a malignant process. An antecolic, side to side cystojejunostomy was made with the use of the Roux-en-Y principle. The antiperistaltic jejunal limb was 12 inches in length. The surgical procedure employed in this case has been reported previously.[12]

Biopsy of the wall of the cyst showed fibrous connective tissue. The amylase content of the fluid from the pancreatic pseudocyst subsequently was reported as 1868 units, and the amylase content of the ascitic fluid 221 units.

Preoperatively, the patient ran a low grade fever up to 100 F, and this persisted for about two weeks. On the fourth postoperative day 1400 cc. of fluid was removed by thoracentesis. The ascites appeared to be recurring, but on April 13 it began to subside and soon thereafter completely disappeared. On April 13 the bromsulphalein liver excretion test was 0.2 mg. per cent, and the thymol turbidity was 2 units. The stools were bulky, soft and malodor-

TABLE 2

Character of pleural effusion

	Ascitic Fluid						Pleural Fluid							Serum Protein		
Date	Amt. (cc.)	Sp. gr.	Total protein (Mg./100 cc.)	Culture	Other Charact.	Amylase (units) per cent	Amt. (cc.)	Sp. gr.	Total protein (Mg./100 cc.)	Culture	Other Char.	Amylase (units) per cent	Serum Amylase (units) per cent	TSP (Gm %)	Alb. (Gm %)	Glob. (Gm %)
3-17-54							1950	1.016	4.3	neg.	lime colored; sl. turbid cell block suspicious of malignancy					
3-19-54	3100	1.015	4.0	neg.	straw colored											
3-20-54	1200	1.024	3.6				950	1.016	4.2	neg.	straw colored			5.2	3.3	1.9
4-3-54	2700		4.5	neg.	creamy light green colored								172			
4-6-54	2500-3000			neg.	cell block neg., cloudy, turbid	221										
4-8-54													97 (Operation: Internal drainage of pseudocyst; amylase content 1868 units)			
4-10-54							1400		2.9	neg.		276				
4-13-54												253	163	5.7	3.0	2.7
4-16-54							1000	1.011		neg.	cell block neg.		186			
4-17-54							900									
4-22-54							950					259		5.2	3.7	1.5
5-3-54										neg.						
5-6-54													461	6.8	4.0	2.8

Date							
5-6-54							
5-11-54	800					244	275
5-16-54							299
5-18-54							213
5-26-54							106
6-2-54							159
6-12-54							150
6-15-54	2100	1.021	4.6			293	
6-16-54	1260	1.020				262	
6-18-54	1500		3.4	neg.		217	32
6-22-54							133
6-23-54							123
6-24-54							123
6-29-54	1000						
6-30-54						198	98
7-2-54	200		2.5		fat 0.6 gm/100 cc. Na 140.1, K 4.2 mg./100 cc.		
7-7-54							
7-14-54							70
7-20-54							51

* Normal range 20–50.

Fig. 3. Barium enema. Depression of the distal portion of the transverse colon is shown. The crescent of air about the right kidney represents previous retroperitoneal air study.

ous. The fat content of the stool on April 23 was 8.6 per cent of dry weight and 1.7 per cent of wet weight. After the patient was placed on pancreatin and a fat saponifying agent, the stools became normal.

Ten thoracenteses were done postoperatively, with removal of a total of 11,110 cc. of fluid. The character of the pleural effusion and its amylase content, as well as the serum amylase levels and characteristics of the ascitic fluid, are noted in table 2. The levels of amylase in the serum and in the pleural effusion are demonstrated in figure 6. Amylase determinations were obtained by the method of Wohlgemuth as described by Bray[3]; it is reported to give a normal range of serum amylase concentration of 15 to 40 units (mg. per 100 cc. of glucose formed by incubation of serum with starch). A bilateral inguinal herniorrhaphy was done on July 13, and the patient was discharged from the hospital on July 29.

Multiple follow-up roentgenograms of the chest showed varying degrees of right pleural effusion, but at no time was there evidence of any underlying cause. A chest roentgenogram on May 4 showed pleural reaction with a small quantity of fluid at the right costophrenic angle. Chest roentgenograms on September 11 and on November 17 (fig. 7) showed residual pleural thickening but no evidence of recurrence of pleural effusion. One year after operation there was no recurrence of the cyst or of the hydrothorax.

FIG. 4. Upper gastrointestinal series. Depression and ironing out of the pars media and antrum are noted.

FIG. 5. Upper gastrointestinal series. Anterior displacement of stomach and pressure defect of the greater fornix of the bulb are noted. In addition, a single calcific nodule is seen within the mass.
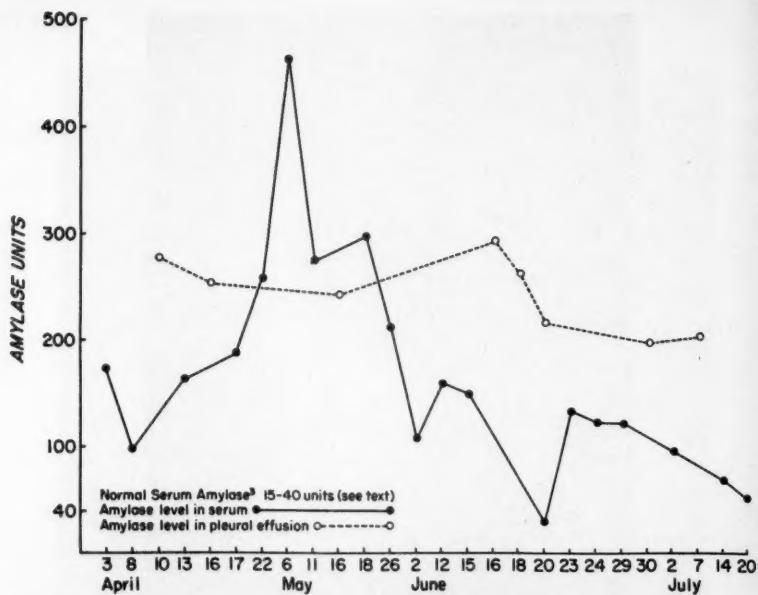
FIG. 6. Amylase levels in serum and in pleural fluid during a period of three and one-half months.
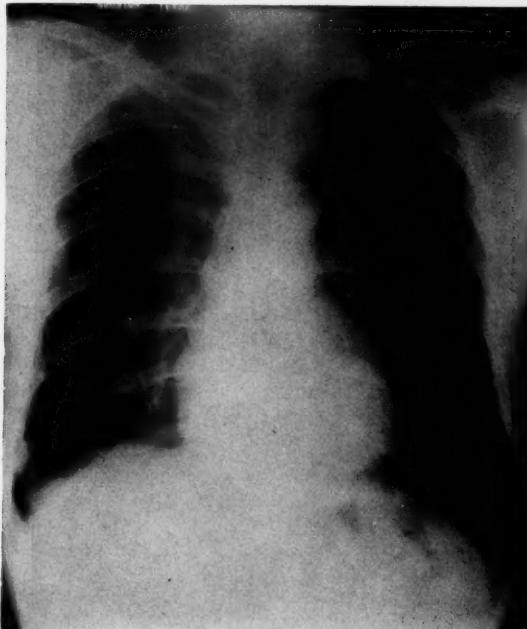

FIG. 7. Upright posteroanterior film of chest (Nov. 17, 1953), showing residual pleural thickening, right costophrenic angle, without evidence of fluid.

DISCUSSION

Hardy[3] classified intra-abdominal diseases causing hydrothorax into two broad categories. The first contains a variety of conditions, such as subphrenic abscess, perinephric inflammation, and pancreatitis, that give rise to inflammation in the immediate vicinity of the diaphragm. The second category embraces a group of cases in which no intra-abdominal inflammatory lesion exists, but in which ascites invariably is present, as exemplified by fibroma of the ovary with ascites and hydrothorax. We believe that hydrothorax associated with pseudocyst of the pancreas should be classified in the first category of intra-abdominal diseases causing hydrothorax.

The mechanism of absorption of fluids and particulate matter from the peritoneal cavity, with reference especially to the diaphragm and its lymphatic vessels, has been studied in detail by many investigators.[4, 6, 9, 11] MacCallum[11] in 1903 was unable to confirm Virchow's work which suggested the presence of preformed openings between the peritoneal lining and the diaphragmatic lymphatic vessels. Instead, MacCallum considered the individual cells in the diaphragmatic peritoneum to be separable by adequate forces and the pumping action of the diaphragm to be sufficient to force fine granules between them. Disagreeing about the existence of potential openings between cells, Cunningham[4] in 1922 concluded that a large proportion of the particulate matter passes through, and not between, the cells in the course of its absorption into the diaphragmatic lymphatic system from the peritoneal cavity. Florey,[6] on the other hand, concurred with MacCallum and considered it improbable that large particles pass through the protoplasm of cells merely as a method of transport.

With regard to the absorption of solutions from the peritoneal cavity, Cunningham stated that the solutions pass in large part directly into the blood stream and that a small quantity of this material probably is absorbed through the lymphatic vessels. He offered two possible explanations for the mechanism of *the small part of absorption* of isotonic solutions that takes place from the serous cavities and cannot be explained on the basis of the known physical laws governing osmosis and diffusion: (1) that the active force is in some way bound up in the metabolic activities of the mesothelial cells and therefore is controlled by laws similar to those active in other cellular functions, and (2) that the active force is of a mechanical nature, (abdominal pressure or respiratory movement) and that the isotonic solution gradually is forced into the lymphatic system or blood vessels by pressure gradient.

Using as a membrane fresh postmortem specimens of human diaphragm in two instances and freshly killed canine diaphragm in a third, Hardy demonstrated that diffusion of magnesium with the use of a 2 per cent solution of magnesium sulphate was slight through the tendinous part, pronounced through the pleuraperitoneum *sandwich* (composed of pleura and peritoneum that were dissected off their respective sides of the diaphragm and laid together), and absent in the case of the muscular portion of the diaphragm. Hardy favors simple diffusion mediated by the abdomen-thorax pressure gradient to explain pleural effusion of abdominal origin.

Of the 10 cases collected from the literature (table 1) the 2 of Bickford[2] and 1 of Traquair[16] were associated with injuries to abdominal organs other than the pancreas or to the thoracic wall. In these it is difficult to attribute the pleural effusion to the pancreatic pseudocyst alone. In Traquair's patient, who developed a pancreatic pseudocyst, bile peritonitis, chylothorax, and chylous ascites during a 10 month period following a blow to the epigastrium, the left-sided pleural effusion was discovered two months after drainage of the pseudocyst of the pancreas. Granulation tissue, but no pus, was found in the left subphrenic space when it was drained.

The etiology of the effusion in the cases of Alivisatos[1] and of Jones[10] is obvious: in the former, a diaphragmatic hiatus allowing the thoracic and abdominal cavities to communicate, and in the latter, extension of the pseudocyst into the mediastinum through the esophageal hiatus, with subsequent rupture into the right pleural cavity. Edlin[5] considered the effusion in his case of mediastinal pseudocyst of the pancreas to have been caused by the presence of a mild degree of cardiac decompensation or by the pancreatic disease which probably was smoldering under the left diaphragm. Phillips[13] did not offer an explanation for the pleural effusion in his patient, in whom a traumatic pseudocyst of the pancreas developed after he was kicked in the upper abdominal region by a horse. We may assume, however, that the sequence of pathologic events, i.e., rupture of the pancreas by the blow, sealing off of the foramen of Winslow, and collection of the pancreatic secretions in the lesser sac, with development of the pseudocyst, offered opportunity for associated inflammation of the left subphrenic space to develop and that the subsequent effusion was *sympathetic*.

Schieppatti,[14] in his discussion of the pleural complications of acute pancreatitis, reported 1 case of a postpancreatitic pseudocyst with pleural effusion that developed 15 days after onset of the pancreatic symptoms. He gave considerable thought to the mechanism of the pleural effusion in this disease and was able to confirm the work of Korte on diffusion of peripancreatic exudates. Schieppatti injected methylene blue into the posterior portion of the pancreas, placed pressure on the area, and observed that the methylene blue largely went to the left diaphragm. After injecting varying concentrations of trypsin into the dog, cat, and mouse intravenously and into the peripancreatic area, he concluded that the pleural effusion was produced by irritation of the pancreatic exudate, which is rich in trypsin, and that it occurs not only by way of the blood stream but by direct diffusion as well. Gross and his associates considered as the most credible theory concerning formation of the fluid either: (1) passage of an irritative substance (pancreatic ferment) through the diaphragmatic lymphatic system to the pleural cavity, where an irritative pleural effusion is initiated, or (2) transperitoneal passage of ascitic fluid into the pleural cavity through the physiologic diaphragmatic openings.

Werner[17] in 1942 described a case of chronic recurrent pancreatitis, associated with cholecystitis and cholelithiasis and with recurrent hemorrhagic pleural effusion occurring on the left when the pancreatic element of the disease predominated and on the right when the gallbladder infection predominated. The

pleural effusion occurred on the left twice and on the right once, the diastase activity of the pleural fluid being 2,048 units and 16,348 units on the two occasions when it was determined. The blood diastase never was higher than 512 units. Werner subsequently checked the diastase activity of the exudate in 2 patients with postpneumonic effusion, 2 with parapneumonic effusion, 2 with tuberculous effusion, and 1 with hemorrhagic effusion associated with bronchial carcinoma. In all the diastase activity in the effusion was within normal limits, as were simultaneous diastase levels in the blood and urine. On the basis of the anatomic position of the pancreas, Werner concluded that the *transmigratory pleuritis* probably was not by contact, that no peritonitis was present, and that most probably the pancreatic enzymes passed through the lymphatic vessels to the subdiaphragmatic network.

Smith[15] described a patient with a recurrent pseudocyst of the pancreas, who had hemorrhagic ascites and right hemothorax. Initial marsupialization had been done two and one-half years previously. Although he mentioned the work of Werner, Smith offered no explanation for the hemorrhagic pleural effusion in his patient.

In our own patient, who had fibrinous adhesions in the right subdiaphragmatic space and no opening palpable in the right diaphragm, we believe that the inflammation of the subphrenic space gave rise to the initial pleural effusion with a high amylase titer, which, in turn, set up a reactive pleuritis with effusion, the amylase level of which was fairly well maintained by exacerbations of the pancreatitis. After the diagnosis of pseudocyst of the pancreas associated with hydrothorax was established, we obtained, over a period of a three and one-half months, 8 amylase determinations on the fluid from the pleural effusion and 19 serum amylase determinations (fig. 6). The varying elevations of the latter, together with the symptomatology, indicated persistence of the pancreatitis. The amylase levels in the pleural effusion were within a relatively constant range of 200 to 300 units and, with few exceptions, the amylase values in the pleural effusion exceeded those in the serum (fig. 6). The pathologic nature of a pseudocyst of the pancreas and its anatomic boundaries afford considerable opportunity for inflammation of the subphrenic space. The subsequent pleural effusion, particularly on the left, as evidenced by occurrence of effusion on the left in 7 of the 11 cases collected, is a result of diffusion of the peri-inflammatory edema mediated by the abdomen-thorax pressure gradient and aided to some extent by the pumping action of the diaphragm. Hardy has drawn an analogy between this and the fact that subcutaneous edema may accompany deepseated abdominal suppurative processes, the edema in such cases spreading through tissue planes many times thicker than the diaphragm. Excluding the patient of Alivisatos, in whom the right-sided pleural effusion was due to an anatomic defect of the right side of the diaphragm, and the patient of Jones, in whom the right-sided effusion was due to rupture of the mediastinal extension of the pseudocyst through the esophageal hiatus, the pleural effusion appeared on the right side in only 2 of the remaining 9 patients. If the effusion in these 9 patients is attributable to pancreatic disease or injury alone, the incidence of 78 per cent

of left-sided pleural effusion with pseudocyst of the pancreas closely approximates that in the 5 cases of pancreatitis reported by Schieppatti,[14] in 4 of which effusion was on the left side and in 1 of which it was bilateral.

SUMMARY

A new case of pseudocyst of the pancreas associated with hydrothorax has been added to those already reported in the literature, bringing the total to 11 such cases.

Except for a few instances in which an anatomic defect in the diaphragm allows the pleural and peritoneal cavities to communicate, we believe that the very nature of the pathology of a pseudocyst of the pancreas and its anatomic boundaries affords considerable opportunity for inflammation of the subphrenic space and that the subsequent pleural effusion probably is the result of diffusion of inflammatory edema from the subphrenic space, aided to some extent by a decreasing pressure gradient from the abdomen to the thorax and the action of the diaphragm.

Pancreatic disease should be strongly suspected in cases of left-sided pleural effusion associated with obscure upper abdominal disease.

The presence of hydrothorax in a patient with an upper abdominal mass does not unequivocally imply that effusion is attributable to metastatic malignant disease.

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THE PROBLEM OF THERAPY IN VARICOSE VEINS OF THE LOWER EXTREMITIES

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To one who has devoted much interest to varicose veins over the past 30 years considerably more perspective appears necessary in our approach to treatment than is in evidence today. If the current literature coming out of our highest sources is any indication of our present approach to management and treatment, then, to this observer at least, it seems timely that our accrued knowledge and experience be re-examined in terms of what constitutes the essential care necessary to prevent debilitating complications, and to obtain more favorable long-term end results.

PREINJECTION ERA

Prior to the late twenties, varicosities of the lower extremities was an inadequately treated and poorly understood affliction of mankind. Bandaging of legs, advice to remain off of and to elevate legs, was the most one could offer such patients while their condition progressed steadily and unfavorably. I well can remember the frustration of the physician and the discouragement of the patient because of the lack of real help or relief we could bring to this condition so that these victims might be freed of the disability that increasingly threatened their pursuit of a normal, useful life. True, vein stripping was then, as now, an accepted surgical procedure, but was done reluctantly and infrequently as the only means of providing some form of attack, even though the instruments and technics were unsurgical at best, invited complications of infection and interstitial bleeding, required protracted disabling convalescence, and accomplished little or nothing in eventual favorable results. To the younger of you, this may appear as a grossly exaggerated picture, but it probably is within the memory of some that our City Hospital wards were filled with disabling complications of this condition and how these patients plagued us in private practice because we had so little to offer.

INJECTION ERA

It is, therefore, not surprising to note how the injection treatment of varicose veins swept the country beginning in 1928. Here was a relatively safe ambulatory treatment to eliminate these incompetent veins by obliteration and subsequent organization. The surgical department at Washington University, in which clinic I initiated and developed this new treatment, enthusiastically abandoned vein stripping, and varicose veins quickly disappeared from the operating schedules in all our medical centers. The voluminous literature that followed remains as further testimony that in this new approach to treatment we finally could offer real help to vein sufferers. Retrograde pooling and stagnation of blood was

eliminated; complicating infections and ulcerations responded favorably; symptomatic and physiologic improvement was striking; and patients flocked to the clinics and doctors' offices in surprising numbers as the news of good results spread.

More important than all this was the impetus felt by our profession everywhere to renewed interest and study of this condition. The peculiar nature of varicose veins, which makes it a special problem for considerations of therapy, now is sufficiently understood to justify the essential thesis of this presentation: that surgery alone, no matter how radical, is not an acceptable or adequate answer to the therapeutic challenge this condition presents. Only a brief statement of some of the more pertinent characteristics of this condition is permitted for considerations of therapy.

ETIOLOGY

Varicose veins occur in the human being because of an inherent hereditary constitutional weakness in the vein walls of certain individuals to which is added the intravascular stress of hydrostatic pressure in the erect human posture. It is this activating effect of hydrostatic pressure in the erect position which is directly responsible for the onset and unfavorable progress of varicose veins and their complications in the inherently predisposed individual. It is for this reason that all harmful influence of their presence can be eliminated as a temporary measure by simple elevation of the extremity above the heart level, or, to lesser degrees, by elastic bandage collapse.

CLINICAL PATHOLOGY AND PHYSIOLOGY

The great saphenous is the longest vein in the body; is subjected to the greatest dependent hydrostatic pressure; is the most poorly supported, and thus usually the first involved. The change from normal to varicosity usually begins in one segment of the thinner walled calf veins. The vein dilates; the associated valve becomes incompetent; the support is removed from under the valves above, and the condition progresses upward until no competent valves remain up to the sapheno-femoral junction. The weight of the column of blood from the heart level to the foot then exerts its greatest hydrostatic pressure into all tributaries with resultant stagnation of blood, or flow in reverse direction. In such individuals, this constitutional weakness in muscle tone or vein wall structure cannot be eliminated by any known means throughout their lifetime. This makes every patient with varicose veins subject to the development of new varices in reverse ratio to how thoroughly any form of therapy may have eliminated all existing varices, and how thoroughly and completely all incompetent feeder veins and incompetent communications with the deep circulation were eliminated at the time of treatment.

Venous pooling and stagnation of blood produces proportionate malnutrition and anoxia of the skin and superficial fascia. In the thin person with no fat between skin and deep fascia, large tortuous sacculated varices may exist for years with minimal soft tissue changes, with only atrophy and pigmentation of the

skin of the lower half of the leg and minimal symptoms of fatigue and aching on standing. In others with much superficial fat, the tissues become indurated by diffuse fibrosis with varicosed channels deeply imbedded, more palpable than visible. The progressive fibrosis of the fatty superficial fascia further impairs local arterial circulation with eventual ulceration necrosis following the most minor or trivial trauma. It is surprising to observe how often long advanced impaired nutrition of the surface tissues may remain subclinical with the patient totally unaware of the impending hazard, with neglect by habit of a seemingly innocent, superimposed trauma, only to have such patients first appear for treatment with the advanced complications of ulceration, cellulitis, or acute thrombophlebitis.

Edema is never caused by, or associated with, simple varicose veins, and can be totally absent in the most advanced presence of this condition. Edema always is the signal of damaging complications and represents lymphatic obstruction as a result of slow encircling superficial fibrosis, or congestive bacterial cellulitis. The presence of edema will vitiate any effort at treatment of the varicose veins or their acute and chronic complications. Therefore, prompt steps should be taken first to eliminate edema by postural drainage and *up-side-down* bicycling exercises, while therapy is directed at elimination of all bacterial activity. If this is not followed by complete elimination of all superficial varicosed systems, the secondary complications quickly recur and progress at a rapid, unfavorable pace with increasing degrees of residual permanent soft tissue damage.

TESTS

Of the tests described for varicosities, the simple rubber tube tourniquet supplies all the information we need to know for treatment purposes. By this means it can be determined quickly if the incompetent valves extend to the sapheno-femoral junction, or through communicating veins to the deep circulation.

DEEP THROMBOPHLEBITIS

Tests for the patency or incompetency of deep veins seldom become a practical consideration in patients seeking treatment for uncomplicated varicose veins. Only if a history of deep thrombophlebitis, or *milk leg*, is obtained or suspected does one need to be concerned significantly about the functional status of the deep venous circulation. The superficial varices which remain as an aftermath of this condition invariably are proved to be unfavorable to circulatory function by the simple test of elastic bandage collapse of these varices with the patient walking. The absence of unfavorable symptoms on such testing indicates the wisdom of eliminating such varices. These must not be mistakenly considered as essential collateral circulation after deep thrombophlebitis, since they are as incompetent in the upright position as any other varices. If it were not for the recanalization of the involved postphlebitic superficial and deep veins, most, if not all, of the postphlebitic complications would be avoided. However, recanalization unfortunately is the rule, leaving them rigid and incompetent. This

situation, through hydrostatic influences, increases superficial and deep varix formations which add to the unfavorable balance between the effective intact collateral return circulation and the incompetent reverse venous flow. If this is allowed to progress unchecked, an intolerable state is reached incompatible with comfort and health of the lower extremity in the upright position. Therefore, real improvement is afforded, functioning collateral veins protected, and complications minimized, through early elimination of much of this reverse load by ligation of the superficial femoral or popliteal veins as indicated, and elimination of the superficial varices as they occur.

BASIC PRINCIPLE OF ALL THERAPY

The one basic principle in the therapy of all varicose veins must be kept in mind here, even more so than in dealing with lesser forms of venous insufficiency, namely, that any benefit from therapy must depend upon the existence of an adequate venous return in the upright position through uninvolved channels so that these may function more effectively after the incompetent reverse venous flow has been decreased or eliminated. It is this single effect that justifies all treatment, and demands that all treatment be as thorough as possible at any stage of varicosity development. Fortunately, ample uninvolved competent collaterals are present in most instances which survive even the more severe deep phlebitic processes. In any case, the future health and well being of such extremities depend upon protection of these essential collaterals against further damage and incompetency, first by early arrest and elimination of the active infection, followed by an early elimination of the compromising and progressively destructive reverse blood flow in the incompetent veins. The goal of any therapy therefore must be the prompt complete elimination of all reverse blood flow in the upright position so that blood moves normally into and out of the extremity.

SCLEROSING INJECTIONS

Under readily controllable conditions the injection treatment has remained, through the years, the only available method which can approach effectively this goal by permanently eliminating any and every varicosity regardless of size, location or condition. It can become tedious for the operator, and requires patience and meticulous thoroughness. For the patient it means safe, ambulatory, economical permanent disappearance of all existing varices with thoroughly satisfying symptomatic and physiologic results.

Originally, sodium salicylate was a sufficiently effective permanent sclerosing agent. However, momentary severe pain after injection caused many workers to turn to other agents of lesser effectiveness. These soon proved unsatisfactory, allowing recanalization and recurrences of treated veins at a rapid rate, or causing perivascular edema which was painful for many days and prevented firm organizing thrombus formation. This relatively unfavorable experience with painful sodium salicylate on the one hand, and a high recurrence rate with lesser agents on the other, led many to abandon treatment through early prejudice against it. As more patients sought treatment in general, they were increasingly

advised against the injection treatment as being of no permanent value. The question might be asked here if this was the way the stage was set for the present revival of vein stripping. If so, I am convinced that we have been insufficiently critical of why this shift has occurred away from injection therapy to the greater emphasis on surgery only.

In spite of postinjection pain this observer successfully persisted in using sodium salicylate until Sotradecol became available. Sotradecol is painless on injection, produces consistent intimal destruction, a firm organizing clot, and minimal perivascular edema. End results in permanent obliteration are dependably consistent. It was soon observed in patients treated by injection alone, that recanalization was prone to occur with more rapid formation of new varices if incompetent communication between the varix and the deep circulation existed at the sapheno-femoral junction or below. Therefore, high saphenous ligation plus ligation of any incompetent communicating veins have been added to the essential treatment as the only surgical procedure which is ever necessary. This usually requires only overnight hospitalization. High ligation with vein transection must be flush with the femoral vein after all regional tributaries are severed and ligated. A portion of the distal segment of the saphenous vein is excised and 3 per cent Sotradecol solution is injected retrograde into the great saphenous trunk when the latter is the principle typical single retrograde channel. When findings are atypical or anomalous retrograde injection is omitted. Too often patients are seen in whom this operation was not done accurately or thoroughly or was not followed by complete sclerosing therapy, whose varicosities had soon recurred or persisted as severely as before, with only an operation scar to indicate any attempt at treatment.

TREATMENT

For treatment purposes, then, varicosities may be classed in two categories. The first includes all early localized varices with no associated incompetent communicating veins from above down, or from within out. Such can be eliminated quickly and permanently by one or two simple office injections as they appear. In so doing, all chance of injury to the extremity from either the disease or the treatment is prevented. Periodic check ups are required for treatment of new varices as they are almost sure to form. All other varicosities fall into the second category, namely, those associated with one or more incompetent communicating veins. After such communication is surgically eliminated, all existing varices are automatically converted to the same character as those in the first category, and are just as amenable to permanent sclerosing cure without vein stripping. After injection of large, bulging varices, a 1 inch sponge rubber pad compressed over the treated veins with an Ace elastic bandage reduces the size of the thrombus mass and the time required for its complete organization.

DISCUSSION

In drawing conclusions for therapy purposes, all that has been stated leads to one pertinent question as I attempt to understand this problem of treatment. Why has vein stripping returned as the popularly heralded and accepted treat-

ment by our profession as evidenced by practically all recent literature on the subject and as evidenced by the operating schedules throughout the country? Experience has proved that the far advanced, thin walled, extremely tortuous or canalizing varix cannot be stripped by any method. These can be just as permanently eradicated by sclerosing injections as can other varices, and must be so treated if they are treated at all. Then why is vein stripping ever indicated or accepted as a method of choice for other lesser forms of varicosities, which respond even more readily to sclerosing injections? Vein stripping not only is unnecessary to obtain a permanent cure of existing varices, but it is a traumatizing radical procedure, unsurgical at best, involving tissues which are already impaired in health, and which places unwarranted emphasis upon the surgical to the neglect of the equally important injection method of treatment.

When it is realized that most surgeons who are stripping veins today had little interest in, and never treated this condition before vein stripping was revived and again made popular, it would appear that just another operation has been added to the operating schedule through blind acceptance, in the belief that a single radical surgical attack could be of real, lasting, therapeutic value. The one inescapable fact that apparently has been lost sight of is that no patient can be cured of varicose veins in the true medical sense of the word "*cure*", no matter how radical the surgery. Then why is a radical procedure revived and practiced as a treatment of choice when no cure is effected thereby? This apparently has happened while more effective available methods for the control of this condition are neglected or excluded. Isn't it timely that we, as surgeons, recognize that everything of benefit that a patient might need of us, need not necessarily happen in an operating room; that also we must be willing to devote some of our attention, interest and efforts to the smaller tasks and more troublesome details demanded by the conditions we undertake to treat? Any failure in the popular practices of our profession to provide the best long term benefits will bring only discouragement again to these patients, and discredit to all treatment. This, in my opinion, would be a step backward three decades to the era before injection therapy.

To what, then, does the summation of our experience point as constituting a positive, responsible approach to this problem of therapy? Because varicose veins are a common, disabling malady affecting a great percentage of the population, and because they are seen by almost every doctor, it appears that our attitudes and understanding can be better organized and mobilized into more effective control of this condition than is evident at present. The most appalling thing to many of us is the vast number of varicose vein victims among us today who present advanced stages of this condition, whose varicosities have existed for tens of years, and who have never been advised to be rid of them by any physician who had observed them. These patients could have been spared great expense, long suffering disability and residual permanent soft tissue injury, if their early localized varices had been eliminated by simple injection when they were first noted on physical examination, and then later as new ones may have formed again. The predisposed individual can be significantly recognized, and

by use of injection therapy can be completely protected against any progress of this condition, thus preventing debilitating complications. This is a worthy and attainable goal which challenges our responsibility to the many victims of this malady.

More often than not, the physician has been regarding this condition as too common and trivial to be significant, and has tended to agree with the patient not to consider treatment until the varices become worse or bother him. By the very nature of their origin, varicose veins always grow worse, both in size and number, and, except in the neurotic, unfortunately never bother the ordinary person to the point of seeking treatment. By the time the varicosities cause symptoms it is rather the complications with varying degrees of permanent soft tissue damage that actually brings the patient to seek help.

The insidious progressive character of varicose veins from the simplest beginnings and the unpredictable onset of complications, makes it incumbent upon the medical profession to explain the character of this malady to patients, and to urge treatment to eliminate all varicosities at any stage of development whenever discovered regardless of lack of symptoms. This responsibility lies heaviest upon the general practitioner whose privilege it is to observe the early presence of varicosities among the populace. However, it also becomes our responsibility in the surgical profession to practice thoroughly effective therapy when surgery is indicated, keeping such surgery to an indicated minimum free of vein stripping, and regarding treatment as incomplete until the last incompetent varix has been obliterated by sclerosing injection.

The nature of this malady in its present state of relative neglect makes it our further responsibility to place greater emphasis upon an understanding of this subject in our medical schools and in our post-graduate contacts with general practitioners everywhere to the end that patients and their family doctors alike may appreciate the great importance of early injection treatment and periodic check ups before symptoms develop, and before surgery becomes a necessity. There is no reason why the general practitioner cannot control this malady by early injection therapy if he has had adequate clinical undergraduate training, and understands when surgery must precede injections in order to be successful. Such a program eventually would go far toward eliminating advanced varicosities and their disabling complications from our medical experience.

As the general practitioner and surgeon of tomorrow, the intern of today, however, sees only the surgical attack on varicose veins and has no knowledge of, or experience with, the injection method of therapy for the reason that it is not a hospital procedure, and because this procedure is no longer practiced in most of the out-patient medical school clinics. This apparently has happened as a result of higher specialization in the rapidly expanding fields of operative surgery within our medical schools, with increasing emphasis upon purely operating room surgical procedures to the rather general neglect of training in the technics and indications of minor procedures which comprise a far greater part of, and which serve a more frequent need for our surgical services in private practice. As such institutional trends pertain to this particular condition, at least,

our responsible care to these victims has been compromised to a serious degree through the neglect of an essential means of therapy which leaves no effective substitute at our command in its control. Therefore, the importance of injection therapy in this picture must be actively revived and again made a part of undergraduate training in our medical schools while it is actively appreciated and practiced by our surgical profession before this attainable goal can be realized.

SUMMARY

It has been emphasized that no patient is ever cured of varicose veins regardless of the therapy employed. The background of our knowledge and of our experience with varicose veins is reviewed. Effective treatment is outlined to meet the needs of this condition as we know them, consisting of sclerosing injections alone, or ligations followed by injections where incompetent communicating veins exist.

It is not enough for the patient to be sent to a surgeon for vein stripping when this condition is encountered.

A plea is made, as supported by an understanding of the character of this condition, that vein stripping be abandoned as an unnecessary radical procedure.

A plea also is made that we meet our responsibility to medical students and to our colleagues in general practice by re-emphasizing the most important place injection therapy holds as a simple effective economic means of controlling the progress and complications inherent in this malady.

Second only to the character of the therapy itself, long-term success or failure in our treatment of varicose veins hinges preeminently on an appreciation of the chronic recurrent tendency of this condition both by surgeon and patient, to the end that neither surgery alone nor injections alone can be considered the answer to this problem, and so that the patient may cooperate with greater understanding in full acceptance of the total treatment required to render him as free and safe from varicose veins and their complications as is possible throughout his life time.

ACUTE POLIOMYELITIS: A FOUR YEAR ANALYSIS IN AN ENDEMIC AREA

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This paper is a study of 800 cases of acute poliomyelitis treated at the Robert B. Green City-County Hospital in San Antonio, Texas from January 1950 to January 1954. The treatment of these patients from the acute phase throughout the convalescent or chronic stage has been directed almost entirely by orthopedic surgeons. This is unique in that pediatricians, internists, and physiatrists usually occupy the dominant therapeutic role during the acute and subacute phases of the disease in most treatment centers in the United States today.

Paralytic poliomyelitis comprised 69 per cent of this series. Thirty-one per cent of the patients were nonparalytic and remained so on follow-up examination. Two per cent of the entire series were initially thought to be nonparalytic throughout their hospital stay, but later were found to have residual paralysis when seen in the out-patient department. These percentages compare favorably with those reported in other large series. Wrong diagnoses were made in 1.3 per cent of patients. This figure is comparatively low because these were patients who were discharged from the hospital with a diagnosis of poliomyelitis subsequently proved to be erroneous. Nonpoliomyelitis patients discovered in the course of hospitalization were transferred to other services, and hence, are not included in these statistics.

Of the paralytic patients, 73 per cent were of the pure spinal type, 7 per cent were pure bulbar, and 8 per cent were a combination or bulbar-spinal without respiratory involvement. Twelve per cent of our paralytic patients had respiratory involvement requiring the use of a respirator. The respiratory paralytic patients were further subdivided as follows: spinal respiratory, 7 per cent; bulbar respiratory, 1 per cent; spinal bulbar respiratory, 4 per cent.

It was surprising to the authors to find that 38.5 per cent of this entire group of patients had polio insurance or had policies in which poliomyelitis was underwritten. Actually, the percentage of patients covered by insurance was much higher in 1952 and 1953 than in preceding years. This fact indicates either greater availability of polio insurance in more recent years or a realization by the public of the prevalence of this disease in this area. The financial responsibility for the remaining patients not covered by insurance, 61.5 per cent, of the total group, was assumed entirely by the National Foundation for Infantile Paralysis.

TABLE I

Paralytics	69%
Nonparalytics	31%
Inconstant nonparalytics	2%
Wrong diagnosis	1.3%

TABLE II

Spinal: 73%
Bulbar: 7%
   Bulbar Spinal: 8%
Respiratory: 12%
   Spinal-Respiratory: 7%
   Bulbar-Respiratory: 1%
   Spinal-Bulbar-Respiratory: 4%

An analysis of the symptoms, signs, and laboratory findings was made in this series with the hope that information of prognostic significance might be obtained. The authors were unable to find any factors in this study to indicate that the symptomatology, physical signs, or laboratory findings at the time of admission were significant in predicting the ultimate degree of involvement or paralysis. Headache, fever, nausea and vomiting, and pain were the most common symptoms noted. These complaints most commonly existed from two to seven days prior to admission. We further noted that there was no relation between the duration of symptoms to the onset of paralysis and the ultimate prognosis. Nuchal or spinal rigidity, muscle spasm and tenderness, areflexia, involving both deep tendon and superficial reflexes, and flaccid paralysis were the most frequent physical signs of diagnostic importance upon admission. We find that the characteristic tripod position when the patient attempts to sit, hamstring tightness or spasm, and the so-called head drop test are the cardinal diagnostic signs of poliomyelitis in our experience. No correlation could be established between the absence of deep tendon reflexes and the ultimate degree of muscular paralysis in an extremity. Hyperactive deep tendon reflexes frequently were observed in the acute phases of the disease. Spinal fluid cell counts in this series ranged from zero to 920 cells. The average cell count approximated 100 per cubic millimeter with a predominance of lymphocytes, although polymorphonuclear leukocytes frequently predominate in the very acute stage of the disease. Cell counts in the paralytic patients were compared with those of consistent nonparalytic patients and found to be essentially the same. It has been our impression clinically that a low spinal fluid cell count in a severely involved patient was a grave prognostic sign, but this could not be statistically substantiated. The spinal fluid protein, sugar, and chloride values appeared to be of significance only in differential diagnosis.

The management of patients with respiratory involvement requires special mention. Great advancements have been made in this phase of treatment in the past 10 years. Among these, have been the development of intermittent positive

TABLE III

Symptoms: Headache, fever, nausea & vomiting, pain.
Signs: Nuchal & spinal rigidity, tenderness & spasm, areflexia, flaccid paralysis.
Laboratory: Cerebrospinal Count

$$\text{Paralytics} \begin{cases} 39\% > \\ 61\% < \end{cases} 100 \text{ cells} \begin{cases} >46\% \\ <54\% \end{cases} \text{Nonparalytics}$$

pressure technics, concept of early tracheotomy, the rocking bed, and the portable chest respirator. However, basic problems of cardiac and respiratory physiology in these patients still is unsolved and continues to be a source of persistent investigation.

Mortality rates in poliomyelitis is confined almost entirely to this group. In the decade of 1920 to 1930, mortality rates approaching 100 per cent in respiratory patients were acceptable. In succeeding decades, the mortality rate has gradually decreased due to the universal adoption of artificial respiration with the *iron lung*. The death rate reported in most treatment centers in the United States is approximately 10 per cent. There were 40 deaths in the present series. All of these patients had respiratory paralysis. This is a mortality rate of 5 per cent.

Our policy in management of these patients with respiratory involvement has been that of early respiratory assistance, utilization of positive pressure, and more recently, early tracheotomy. It cannot be emphasized too strongly the great importance of frequent determinations of tidal air, vital capacity, blood pressure, pulse and respiratory rates in the early hours of admission. Far too frequently, these patients are not placed in a respirator until frank cyanosis and circulatory collapse appear. When the pulmonary and cardiac reserves have been exceeded in this manner, the prognosis is grave and frequently fatal. This was further emphasized to us by reviewing the records of deaths occurring in this series. We believe that early, elective tracheostomy is a great adjunct in the management of these patients both in the maintenance of an open airway and as the most efficient means of administering positive pressure. The mortality rate in this series has been definitely decreased in the past two years since this policy has been enforced. Tracheotomies in these patients should be done at the level of the second cartilage ring because of easier management in the respirator. We believe that positive pressure, combined with tracheotomy, is a distinct advantage by producing better and more efficient pulmonary ventilation and definitely decreases the incidence of atelectasis, pulmonary edema, and bronchopneumonia, the most frequent complications in respiratory patients. Bronchoscopy should be done at the time of tracheotomy through the tracheal opening so that the pulmonary tree is free of mucous obstructions initially. Repeated bronchoscopies frequently have been necessary in this series.

In regard to the degree of initial involvement and the final or ultimate paralysis, no distinct correlation can be made from this study. In general, however, our experience has been identical with others in that muscles testing poor or less

initially usually show marked residual paralysis at the end of one year. On the other hand, muscles testing fair plus in the acute and early convalescence stages almost always return to good or normal function. An attempt was made in this series to grade extremities as a whole according to the degree of motor paralysis, but we have found such attempts to be entirely impractical. Similar gradings and tables have been devised by Green and others. The fallacy of extremity gradings is failure to recognize that certain muscles or muscle groups are of greater functional importance than others. For example, we have found that paralysis in the gluteus medius-minimus complex, gastrocnemius, or deltoids may seriously limit the function in an extremity where the remaining musculature is essentially normal.

In the acute stage, treatment in this series consisted primarily of symptomatic therapy. Hot packs and heat were administered to those patients exhibiting spasm and pain. Various splinting devices and rolls were utilized for immobilization in functional positions. Extremities were put through a passive range of motion as soon as pain and spasm subsided. Most of this treatment was done by nurses and aides on the ward. Treatment by trained physical therapists was minimal in this stage. The average duration of hospitalization in this series was 17.9 days.

Convalescent and chronic patients were followed in the out-patient department. Orthodox treatment of bracing and out-patient physical therapy was prescribed for those patients with residual paralysis. It has been our policy to initially over-brace the severely involved patients with gradual reduction of such appliances. We have been impressed with the inadequacy of accepted types of bracing in patients with severe involvement of the hip abductors and extensive lower extremity paralyses. The authors believe that the Bennett method of bracing these patients with bilateral long leg braces, abdominal corset, and crutches, without protection of the flail hips, is to be strongly condemned. The use of spinal extension bracing, utilizing a chair back brace, wide abdominal apron, and connected to the long leg braces by a drop lock is a much more efficient arrangement and prevents the frequent complications of pelvic obliquity, lumbar lordosis, and subluxations of the hips so frequently seen in this situation.

Fifty per cent of the patients in this series are known to have completely recovered from their illness without any evidence of residual paralysis. Thirty-five per cent of the patients have been lost to follow-up which is a regrettable figure and demands explanation. Most of the paralytic patients with polio insurance are being treated by private physicians and accurate follow-up information on these patients is unavailable at this time. A fair number of nonresident patients are being treated by their local physicians. It also should be remembered that

TABLE IV

Complete recovery	50% of entire series
Lost to follow-up	35% of entire series
Operated upon	3% of paralytic group
Surgery required in future	35% of paralytic group

the Latin-American population is high in this area and these patients frequently are reticent to accept proposed medical treatment and advice. Three per cent of the paralytic patients in this series have already had surgical procedures. We have estimated that 35 per cent of the entire group of paralytic patients will require some type of reconstructive surgery in the future.

SUMMARY

Eight hundred cases of acute poliomyelitis seen in a treatment center have been analyzed. Sixty-nine per cent of these patients were paralytic and 31 per cent were nonparalytic. An erroneous diagnosis was made in 1.3 per cent. The mortality rate in this series was 5 per cent. No correlation between the symptoms, signs, and laboratory findings on admission and the ultimate muscular involvement could be established. Early use of the respirator, positive pressure, and elective tracheotomy is advocated. Patients with severe initial paralysis showed poor functional rehabilitation while those with fair or better muscles initially showed the greatest degrees of functional return. Fifty per cent of the patients in this series demonstrated complete functional recovery without residual paralysis.

SURGICAL TECHNIC

MANAGEMENT OF THE DUODENAL STUMP

A RATIONAL TECHNIC FOR DIFFICULT CASES

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Serious complications and death following gastric resection of the Billroth II type for duodenal ulcer are almost directly parallel to the complications and accidents associated with management of the duodenal stump[1]. Welch[5] wrote that success or failure of gastrectomy for duodenal ulcer depends on the handling of the duodenal stump. Waugh[4] stated that this complication alone creates a mortality rate of 2.5 per cent for gastric resection. The importance of the management of the duodenal stump prompts this writer to relate important observations in the performing of over 100 gastrectomies (Billroth II type) and to suggest a rational technic that may be used in difficult cases.

In general a safe closure usually can be made if one carefully appraises the pathology present and proceeds with a closure applicable to the pathology using sound surgical principles, and then doing an anastomosis that will least likely result in increased afferent loop pressure.

PATHOLOGY ENCOUNTERED

Recognizing the type of pathology that is present and orienting one's self as to important neighboring structures is most important, for in so doing the surgeon may determine the type of procedure that is applicable and possible under the conditions present. Pathologic changes produced by duodenal ulcer may be entirely limited to the duodenal wall; however, these changes frequently extend beyond the duodenum producing a varied and complicated picture on opening the abdomen.

Ulcers may be found in any portion of the duodenum. They usually are found proximal to the descending duodenum and most frequently within 2 cm. of the pylorus. They may be located on either wall or border, and may extend from one wall to a border and even around to the opposite wall involving almost the entire circumference of the duodenum. They occasionally are multiple. Repeated recurrence of inflammation leaves scarring and cicatrization with resultant stenosis and shortening and possible formation of secondary diverticuli proximal to the stenosis, or an outpouching on the opposite wall or border from the scarring. Extension of the ulceration through the duodenal wall, subacute perforations and acute perforations, sometimes leads to involvement and attachment to adjacent organs such as the pancreas, common duct, gallbladder, liver, mesocolon and colon. Extra-duodenal adhesions vary in extent and may cause kinking or

deformity of the duodenum and attachment to surrounding organs in abnormal positions. Occasionally one may be called upon to do a gastric resection and a duodenal closure in a patient who has had a previous gastro-enterostomy with marginal ulcer and possibly a gastro-jejuno-colic fistula.

The duration of the pathologic process may vary considerably and may be of different stages in the same patient. One may see a localized perforation with a thick edematous wall in an old scarred duodenum. This is important, because very often one cannot tell how friable the duodenum will be until he has dissected it free and gone too far for an alternative procedure. Recognizing that this type of friable duodenum will not hold sutures or permit the usual inversion procedure may mean the difference between success and failure.

USUAL TECHNIC

Incisions for gastrectomy vary considerably among different surgeons, who may use any one to his liking as long as it gives adequate exposure for handling the duodenum and likewise sufficient exposure for a high proximal gastric resection and anastomosis, or a vagotomy as an alternative procedure. The author uses almost entirely, an upper left rectus, paramedian, muscle splitting incision which gives ample exposure of the duodenum when a self retaining retractor pushes the right edge of the incision back against the right costal margin. The pylorus actually is in the midline with the duodenum only a little to the right and no difficulties of exposure have been encountered even when there are extensive adhesions in this area. The incision on the left facilitates high proximal exploration, including the lower end of the esophagus, high proximal resection and anastomosis and a vagotomy if necessary. The incision facilitates the forming of a closed space around the duodenal stump and a potential external fistula.

On opening the abdomen numerous adhesions may be encountered. After freeing those from the anterior abdominal wall which may be extensive if there has been previous surgery, the lower edge of the liver is freed and likewise the colon which is pushed down out of the way exposing the prepyloric region of the stomach. Folding over of the duodenum or any unusual attachments due to adhesions from extension beyond the duodenal wall, previous perforations and peritonitis should be recognized and freed first. An opening is made in the gastro-colic omentum and a finger is placed beneath the prepyloric portion of the stomach which is raised and placed under tension. The right gastro-epiploic vessels are divided about 2 cm. proximal to the pylorus and then the dissection of the inferior border of the stomach and duodenum is later continued next to the wall inside these vessels which with their surrounding omentum will be used to cover the closed duodenal stump. Dissection also is carried as far as possible close to the anterior duodenal wall and then the superior border is freed as well as possible by cutting peritoneal bands and adhesions and the right gastric vessels. When the case is complicated with many adhesions the dissection is carried down a little at a time close to the duodenal wall using fine hemostats. Freeing of the posterior wall may be facilitated by dividing the stomach proximal to the pylorus and rotating it over to the right. Freeing the second portion of the duodenum by

incising the peritoneal reflection when feasible, facilitates suturing the relatively mobile anterior wall to more rigid fixed portions that usually result from posterior perforations into the pancreas. At this time usually one can determine whether the ulcer can be removed with the specimen, cut through, or left in the duodenum.

A small to moderate sized intramural ulcer located close to the pylorus ideally can be removed with the specimen. With adequate length and mobility of the duodenum, closure of the duodenum can be made with three rows of suture. A continuous row of chromic no. 00 catgut suture over a Kocher forceps (Parker-Kerr stitch) or inversion type suture without clamps doubled back in a seromuscular stitch and then covered with another layer of interrupted fine silk, cotton or catgut is applicable to such uncomplicated cases. This rather standard technic, with slight modifications, has been used when possible by the writer and on the Louisiana State University Service at Lafayette Charity Hospital[6].

Even when the ulcer has extended through the duodenal wall and perforated into the pancreas this type of closure still can be used if the ulcer is relatively small and close to the pylorus and adequate length can be freed up distal to the ulcer without encroaching on the common duct region. In these cases the ulcer bed is left in the head of the pancreas and no attempt is made to excise it or cover it with a portion of the duodenum. This area is drained to the outside.

Difficulties arise when the ulcer is more distal to the pylorus and when repeated recurrence of inflammation has caused shortening and either pulling of the common duct and papilla of Vater upward or the pylorus downward. This shortening is practically always more pronounced on one border, thus forming an out-pouching opposite it or a secondary diverticulum proximal to it. These findings should be considered danger signals and, when seen, necessary precautions should be taken. External dissection should be limited when the above condition exists, because one cannot accurately tell the location or extent of the ulceration and its relation to important structures by external palpation. Also, if the ulcer cannot be removed, further external dissection will only loosen the sealed off area around it, damage the blood supply and make a leak more probable. Troublesome hemorrhage and trauma to the pancreas also is a possibility when dissecting under these conditions.

In these cases it is best to transect the duodenum just distal to the pylorus and hold it open with Babcock or Allis forceps. Suction or sponge out the open duodenum and inspect first. Then insert the index finger into the duodenum so that the location and extent of the ulceration, as well as the direction, amount of constriction and shortening of the duodenum, can be determined. Likewise the approximate location of the common duct, papilla and other important structures should be determined. Clamps should not be applied because they devitalize available duodenal wall and make it difficult to suture safely. It is not necessary to remove the duodenal ulcer unless there is acute hemorrhage. One should not be too bold in trying to remove an ulcer when an important structure may be jeopardized. One should utilize all the duodenal wall made available by transecting the duodenum just distal to the pylorus and a closure can be made

whether transverse, vertical or oblique. I have had to trim and shape parts of the duodenal wall to make a snug closure without tension, similar to the way a seamstress trims clothes to make a fit. Sutures may be continuous or interrupted or a combination of both in completing the closure. Sutures may be absorbable or nonabsorbable, but I prefer, at least, catgut as the first layer if more than one layer of sutures can be used. Inversion should be strived for but is not absolutely necessary. Sutures should be placed without tension. It is especially important when the ulcer is left that one should be careful to have adequate length of duodenal wall proximal to it, or at least sufficient opposite duodenal wall that can be folded over to the fixed edge near the ulcer without tension.

The duration of the pathologic process often determines the difficulty that will be encountered, and one should remember that pathologic processes of different duration may be present in the same patient. Occasionally a dissection is started and there is found a subacute perforation or a perforation with a localized abscess or a pocket containing bowel contents around the duodenum. In these cases the duodenal wall is very friable and will not hold sutures. Inversion layer closure is impossible due to the thickened, edematous and friable duodenal wall and only trouble will result if one tries to do the impossible with this type of duodenum.

CASE REPORT

P. W., a white man, aged 47, gave a history of recurrent epigastric pain, usually occurring in the autumn, relieved by taking alka-seltzer and baking soda. He first was seen on Sept. 22, 1950 because alkalis had become ineffective and he had had intermittent vomiting for three days. Examination essentially was negative except for moderate tenderness with slight rigidity on deep pressure in the epigastrium. Roentgenologic studies showed a large ulcer in the pyloric region and marked irregularity in the antral region. The patient refused operation, but returned five days later when the pain became unbearable. Exploration on September 28, showed that the patient had an old perforation into the head of the pancreas and, in addition, an acute perforation which had formed a localized abscess around the duodenum. The old ulcer was broken into while dissecting the prepyloric portion of the stomach. The bed was left on the pancreas and the dissection was continued just distal to the pylorus where the thick edematous duodenum was divided. A very thick and friable duodenal wall was closed with one layer of interrupted fine silk sutures. The closure was covered with omentum and parts of the round ligament which were sutured over the closure. This area was drained to the outside. The patient was discharged from the hospital in 1 week and, other than a wound infection, made an uneventful recovery. When seen 1 month later, he had gained weight and had no complaints referrable to his gastrointestinal tract.

Comment. In the above case the duodenal opening was closed with one layer of interrupted sutures without tension. Inversion was not done because of the thickened and friable duodenal wall. This was somewhat similar to the method used in the closing of a bronchial stump; the remainder of the closure was directed toward buttressing this closure. In this particular case the right margin of the gastro-colic omentum, the round ligament and some of the greater omentum was sutured around the closure in the form of a patch. Obviously rolling the suture line into the adjacent pancreas was out of the question. In this writer's experience an edematous and friable duodenal stump that is difficult to close,
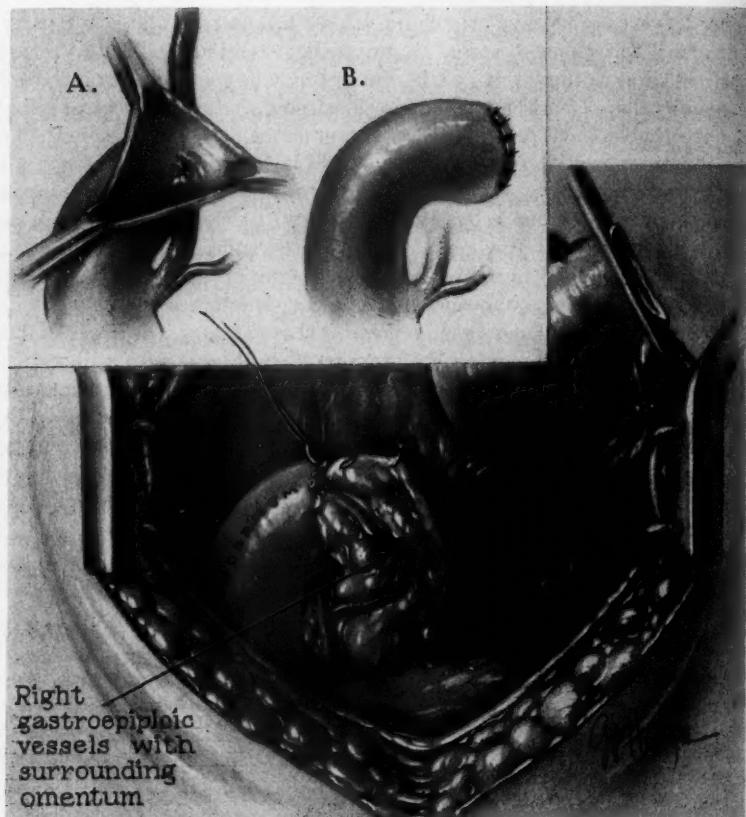
FIG. 1. Insert A. Duodenum after transection just distal to pyloris held open with Babcock forceps showing ulcer in close proximity to common duct. B. Duodenal stump closed with one layer of fine silk sutures. Inversion is not complete. Drawing illustrates right gastroepiploic vessels with surrounding omentum sutured over closed duodenum.

(those you would like to have added protection by covering the suture line) one is not able to bury the suture line in the pancreas[3]. It is better to close the duodenal stump with well placed nonconstricting sutures, even with just one layer, and then place no more sutures in or through the wall. The right vascular margin of the gastro-colic omentum and some of the greater omentum, if necessary, is sutured over the closure.

TECHNIC

The above case and several similar to it led us to devise a safe method for duodenal stump closure, when due to pathologic changes or the location and extent of the ulcer, only one layer of suture could safely be used.

This technic utilizes nature's method of plugging openings in the intestinal

tract, and forms a closed space around the duodenal stump leading to the outside which would result in only a fistulous tract should the stump leak. The vascular right margin of the gastro-colic omentum (right gastro-epiploic vessels and surrounding omentum) is sutured so that it covers the closed duodenal stump and then the lower edge of the falciform ligament is sutured to the duodenum distal to the closure, separating it from the right subphrenic (subhepatic) space. Two penrose drains placed down over the omentum covering the closed stump are brought out along the left side of the falciform ligament through the linea alba. One drain may be made a little longer than the other and placed over to the left in the region of the anastomosis. When a duodenal stump leaks or blows out and there is no way for it to drain to the outside, the consequences are very serious, and often fatal, unless surgical intervention is immediate and then the convalescence is likely to be stormy. It is stated that drainage of the duodenal stump has been one of the most important advances in technic that has caused a decrease in the mortality rate of gastrectomy. We do know that when a duodenal fistula of the end type develops following gastric resection in which drainage of the stump region is employed, that patients usually get well in about 85 per cent of the cases. We also know that in a very difficult stump closure, or those thought to be technically impossible, deliberate formation of an external fistula gives an ultimate favorable result[2]. With this in mind, as complete a closed space as possible is formed around the duodenal stump leading to the outside which is in reality a potential external fistula. This procedure has been used in 16 cases. These have included acute perforations and emergency gastrectomy for massive hemorrhage as well as the usual elective cases.

Probably the most difficult type of case is the perforating posterior duodenal wall ulcer some distance from the pylorus forming an inflammatory hard mass and involving the head of the pancreas, common duct and gallbladder. Some classify this as a type of ulcer not suitable for gastric resection which should be treated by vagotomy with gastro-enterostomy. However, with this treatment it is doubtful that there will be healing of the ulcer and complete relief, when an adjacent organ forms part of the wall of the duodenum.

Very often it is difficult to determine whether the lesion is safely resectable until considerable dissection has been done and the operator has gone too far to turn back. The following case demonstrates this difficult type.

CASE REPORT

H. B., a white man, aged 33, gave a history of a ruptured duodenal ulcer repaired 16 years before. He obtained relief with medical treatment which he followed at intervals since that time. He sought medical aid in May 1954 because the epigastric pain had been very severe for three weeks with occasional vomiting in spite of a rigid diet and all drugs including banthine and even narcotics. Examination showed slight tenderness in the epigastrium and in the upper right rectus scar. Roentgenologic studies made on May 8 showed marked deformity in the pyloric portion of the stomach extending into and including all of the duodenal cap. There was a pseudodiverticulum and 35 per cent gastric residue at 4 hours. Exploration through an upper left rectus incision on May 28 showed many adhesions which had to be freed before exposing the pyloric end of the stomach. Dissection was continued downward through an extensive inflammatory mass extending down and around the

descending duodenum and involving the common duct, gallbladder, and structures in the
region of the foramen of Winslow. As the dissection was continued down close to the duo-
denal wall the whole proximal end of the duodenum suddenly was separated from the large
inflammatory mass. It was found that the wall of the duodenum and diverticulum was made
up of fibrous tissue forming part of a large inflammatory mass and involving the structures
in this area. Mucosa was seen in the depths of this fibrous cavity. A finger placed in the open
end of the duodenum coursed immediately caudad indicating it was the descending duo-
denum. It was obvious that inversion or layer closure would be impossible in this case.
The proximal 1.5 cm of the open duodenum was inspected and palpated for the papilla of
Vater or an accessory pancreatic duct. The mucosa was freed carefully by scissors dissection
over the finger for only a very short distance so as to allow it to be pulled together with one
layer of interrupted sutures. The stump of the right gastroepiploic vessels with its surround-
ing omentum was sutured around the edges of the opening, thus sealing the closure. The
falciform ligament then was sutured to the descending duodenum distal to the closure and
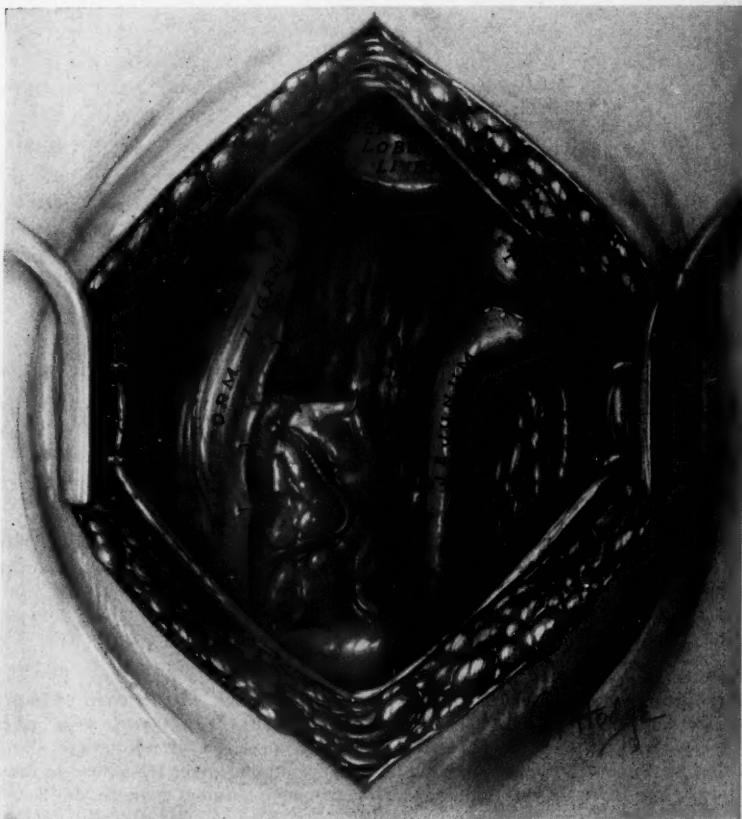
Fig. 2. Falciform ligament sutured distal to closed duodenum separating it from right
subphrenic (subhepatic) space. Closed space bounded below by transverse colon and meso-
colon. Two penrose drains are placed down to and around omentum covering stump and
brought out along the left side of the falciform ligament through the linea alba.

two penrose drains were placed down to and around the omentum covering the closed stump and brought out through the linea alba just to the left of the falciform ligament. An 80 per cent gastric resection, anterior Hofmeister with the proximal loop at the greater curvature, was completed. The patient made an uneventful recovery and was discharged from the hospital on the sixth postoperative day. He has gained 20 pounds since operation (March 1955).

Comment. It is believed that this type of closure can accomplish results with the least danger or damage to the common and pancreatic ducts and other important structures. It demonstrates that by placing only a few nonconstricting interrupted sutures, closure can be accomplished even if only in one layer. Such a closure has a minimum of chance to encroach on the pancreatic ducts and the papilla of Vater, apparently no more danger than catheter duodenostomy which causes considerable morbidity. There was a minimum of trauma to the pancreas. The mucosa only was removed from the portion of duodenum adherent to the pancreas leaving the large ulcer bed and fibrotic mass undisturbed. Choledochostomy with the insertion of a guide in the common duct for excision of the ulcer was not feasible in this case. Such a procedure usually entails more dissection in the head of the pancreas than is desirable. In this case it would have been particularly hazardous because the gallbladder could not definitely be identified and the common duct was intimately incorporated in the inflammatory mass. The closure of the duodenum was covered with the vascular right side of the gastro-colic omentum and also pieces of the right margin of the greater omentum. The drain brought out through the linea alba caused much less post-operative discomfort than a subcostal drain because there is much less motion and this area is relatively devoid of nerves.

SUMMARY

Success of gastrectomy (Billroth II type) for duodenal ulcer depends to a large extent upon proper management of the duodenal stump.

Pathologic changes of duodenal ulcer are discussed briefly with special emphasis on choosing the surgical procedure applicable to the pathology found.

The upper left rectus muscle splitting incision is used because it facilitates high proximal exploration, high proximal resection and anastomosis, and the forming of a closed space around the duodenal stump for a potential external fistula.

More difficult types such as those with shortening of the distance between the pylorus and common duct, or complicated by secondary diverticula, are best treated by early transection of the duodenum just distal to the pylorus. By a combination of intraduodenal and extraduodenal inspection and palpation one can better determine the position of important structures.

A rational technic for the closure of the duodenum in difficult cases is presented. It is applicable in cases with acute inflammatory changes and a friable duodenum, with chronic scarring with shortening, and with posterior perforations into the head of the pancreas. In these cases the duodenum is closed as well as possible without tension even if it is just the mucosa, and only one layer.

This is done with a minimum of dissection so as to preserve blood supply and minimize injury to adjacent important structures. Inversion of the duodenal suture line is not absolutely necessary and no attempt is made to roll the suture line onto the pancreas or cover an ulcer crater left on the pancreas.

The closure of the duodenum is covered with the right gastro-epiploic vessels and the surrounding omentum. A closed space then is formed around the duodenal stump by suturing the falciform ligament to the duodenum distal to the closure. This space is drained to the outside by two penrose drains just to the left of the falciform ligament, thus creating a potential external fistula.

A gastro-jejunal anastomosis that is least likely to cause increased pressure in the duodenum is carefully made.

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MANUAL EFFICIENCY AND TEAMWORK IN SURGERY

A half century ago certain psychologists began the study of motion economy. Methods for improving efficiency in bricklaying by such workers as the Gilbreths, of "Cheaper by the Dozen" fame, laid the groundwork for the next step in the industrial revolution. The assembly line method which made the modern American motor car possible was a definite outgrowth of efficiency methods. Today even the modern kitchen is planned upon these same principles.

In the popular fancy a surgeon is the epitome of manual dexterity. Brilliant technic is thought to guide nearly every move. Although this is true for some outstanding surgical technicians, the average state of affairs differs widely from this ideal. How common it is to see even a successful surgeon of many years experience using at least twice as many motions as necessary to close a wound, for example, although he may have done the operation several thousand times. He wastes his own valuable time; actually adds to the hazard to his patient, and increases appreciably and unnecessarily the already staggering costs of medical care, because operating room costs must be on a time basis. How tragic a contrast it is to see this same surgeon concentrating fiercely on lessons to teach him just how to hold and swing a golf club!

How does this situation come about and what can we do about it?

The very rugged individualism which fosters independence, and leads to progress so often, is here an impediment. It is as though each man were beginning where his ancestors began instead of where they left off. The fear of stereotypy seems to push aside the routine conformity which leads to efficiency in teamwork.

This is in no sense an argument for all surgeons doing the same type of operation and doing it alike but only for uniformity in the window dressing around the small hard core of the surgical procedure itself. A surgeon may resect a carcinoma of the colon in his own inimitable way and be as individual as he likes, but opening the abdomen, getting exposure, achieving hemostasis, and closure will be made up of essentially the same motions and routine steps as used by all other surgeons. The widespread utility of a Ford or Chevrolet depends in no small measure upon the fact that its bolts and nuts are interchangeable with those of other like cars.

The manual superiority of man over monkey is due largely to the opposability of his thumb to his fingers. This basis is the same for surgeon and mechanic. A manual act like the actual performance of surgery must depend for efficiency upon principles which are simple and definite, whether these principles be unconsciously or consciously followed. If they are consciously sought out, studied, and followed, the rewards are outstanding. Here are some of the factors involved:

FACTORS IN INDIVIDUAL EFFICIENCY

1. *Motion economy*—No unnecessary acts—each motion smooth and minimal and contributing definitely to the result—natural and immediate progress into the next sequence.

    a. Tools near at hand—always grouped the same way—most frequently used
       tools nearest.
    b. Motions not repeated—made slowly enough that they always are accurate
       —speed never sought at the expense of errors.
2. *Effort economy*
    a. Larger muscles used for heavier work—smaller (hand) groups saved for
       finer, more accurate work.
    b. Motions made from extreme *towards* neutral positions.
    c. Relaxation when possible—sitting or resting at work.
3. *Use of both hands cultivated.*

FACTORS IN TEAM EFFICIENCY

1. Routine followed—all team members to know it.
2. Everybody always busy—entire job divided as to quality and quantity of
   work to keep each member occupied, but within his ability.
3. Properly timed steps.

Assiduous attention to these principles will decrease the time needed for the
routine parts of surgery for any team, often amazingly. It is in no way belittling
to the judgments of surgeons that these mechanical features be stressed. Indeed
decisiveness as one of the most valuable surgical assets will be most strongly
supported by this approach to technic. The stakes in surgery are far too high
for such an important feature as efficient speed to be neglected.

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BOOK REVIEWS

The editors of The American Surgeon will at all times welcome new books in the field of Surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

The Foundations of Surgery. By GEORGE PERKINS, M.C., M.Ch., F.R.C.S.; Professor of Surgery, London University. Orthopedic Surgeon, St. Thomas's Hospital. E. & S. Livingstone, Ltd., Edinburgh and London, 1954. The Williams & Wilkins Co., Baltimore. Price $3.00.

We frequently are exhorted as students to learn, and as instructors to teach, the "principles of surgery" rather than its technics. These principles, however, seldom are clearly formulated, are not unanimously agreed upon, and, in the usual textbooks of surgery, lie buried under a mass of facts and theories bewildering to the student.

Mr. Perkins, in this very practical primer entitled *The Foundations of Surgery*, has done a creditable job of formulating basic principles using a minimum of well chosen clinical facts. He emphasizes the history, the physical examination, and their logical interpretation as applied to common problems of injury and surgical disease. While the chapter on shock seems to this reviewer to be quite weak, the chapters on injuries seem especially good. While there are some statements of supposed fact with which all will not agree, on the whole the content is accurate and up to date with current advances in surgery. The volume is clearly and interestingly written and small enough so that its complete perusal and assimilation will not seem an impossible task even to the less strongly motivated student. It is recommended not only for the third year student (for whom it was written) but also for the interne and beginning resident as well.

HUGH A. FRANK, M.D.

Fractures in Children. By WALTER PUTNAM BLOUNT, A.B., M.D., F.A.C.S.; Chairman of the Orthopaedic Section, Milwaukee Children's Hospital; Attending Staff Surgeon, Columbia Hospital, Johnson Emergency Hospital, Milwaukee; Consulting Staff, Milwaukee County Hospital; Member of the American Orthopaedic Association, American Academy of Orthopaedic Surgeons, Societe Internationale de Chirurgie Orthopedique et de Traumatologie; Honorary Member, Deutsche Orthopadische Gesellschaft. The Williams & Wilkins Company, Baltimore, 1954. Price $9.50.

Doctor Blount's interest in this field is well known through his instructional courses at the American Academy of Orthopedic Surgery. It has been the hope of all surgeons attending these lectures that he would some day publish in book form his experiences in the management of these fractures. This book more than lives up to expectations. The 260 roentgenograms show valuable experiences gained through 20 years of practice. Seventy-six other figures aid in understanding features of diagnosis and treatment. Certain basic principles are illustrated in cartoon form. This novel manner of presentation makes them easy to remember.

The whole book emphasizes clearly the importance of the closed treatment in the majority of fractures in children. It is particularly applicable at this time when there is a trend in some areas toward more radical measures. This book should be an essential possession of anyone who is interested in the treatment of fractures in children.

LYNN O. LITTON, M.D.

Clinical Neurosurgery. Proceedings of the Congress of Neurological Surgeons, New Orleans, La. Baltimore, the Williams & Wilkins Company, 1955. Price $8.00.

This book is a formal publication of the papers and panel discussions presented at the 1954 annual meeting of the Congress of Neurological Surgeons.

The three recorded papers of Sir Geoffrey Jefferson are adequate justification for pub-

lication of this book. They are concerning integration of the brain, trigeminal neuroma, and chiasmal compression by intracranial aneurysms. Each is presented in magnificent style and is a solid contribution to the neurosurgical literature.

The panel discussions are on the anatomy and physiology of the frontal lobes, psychosurgery, and fluid and electrolytes, and each summarizes quite well the latest concepts on these subjects.

This worthwhile publication as Volume One undoubtedly will start a series of excellent books, and will tend to hold high the standard of papers presented at this Society.

WILLIAM P. WILLIAMSON, M.D.

Surgical Technograms. By F. M. AL AKL, M.D., Associate Attending Surgeon, Kings County Hospital. New York, Toronto, London, McGraw-Hill Book Company, Inc., 1954. Price $12.00.

This book contains 36 descriptions of the operative technic of many of the frequently used operations. It is planned chiefly for use of interns as a concise book of reference to guide them in the technic of the basic operative procedures.

The importance of a knowledge of anatomy is emphasized. A section on anatomy is part of each description of the operation. The line drawing illustrations are many and of good quality. Each step in the operation is adequately described.

For the radical breast operation this author uses the original type of the Halsted incision. The reviewer believes that this incision should be considered obsolete. Any incision for radical mastectomy which may result in a scar extending from the chest wall to the arm across the axilla should not be used. The choice of technic used in thigh amputation may be questioned. The author uses a circular incision made with the old style amputation knife. Long anterior and short posterior flaps, which include all tissues down to the bone, will make better stumps.

Obviously the author has not intended to present a complete book on operative surgery. It would hardly be useful for the resident in surgery, but would help interns and perhaps medical students who are being introduced to surgical technic.

THOMAS G. ORR, SR., M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Local Analgesia: Brachial Plexus. By R. R. MACINTOSH, M.A., D.M., F.R.C.S., D.A., M.D. (hon. causa), Buenos Aires and Aix-Marseilles; Nuffield Professor of Anaesthetics, University of Oxford; and WILLIAM W. MUSHIN, M.A., M.B., B.S. (Lond.), M.R.C.S., F.F.ARCS., D.A., Professor of Anaesthetics, Welsh National School of Medicine, University of Wales. Illustrated by Miss M. McLarty. Third Edition. E. & S. Livingstone, Ltd., Edinburgh and London; Williams & Wilkins Company, Baltimore, Maryland. 1954. Price $3.00.

Ion Exchange and Adsorption Agents in Medicine: The Concept of Intestinal Bionomics. By GUSTAV J. MARTIN, Sc.D., Research Director, National Drug Company. Illustrated with 15 line drawings and 11 photographs. Little, Brown and Company, Boston, Toronto, 1955. Price $7.50.

Early Care of Acute Soft Tissue Injuries. Committee on Trauma. First Edition, 1954. American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.

An Outline of the Treatment of Fractures. By the COMMITTEE ON TRAUMA. Revised and amplified. Fifth Edition, 1954. American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.

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